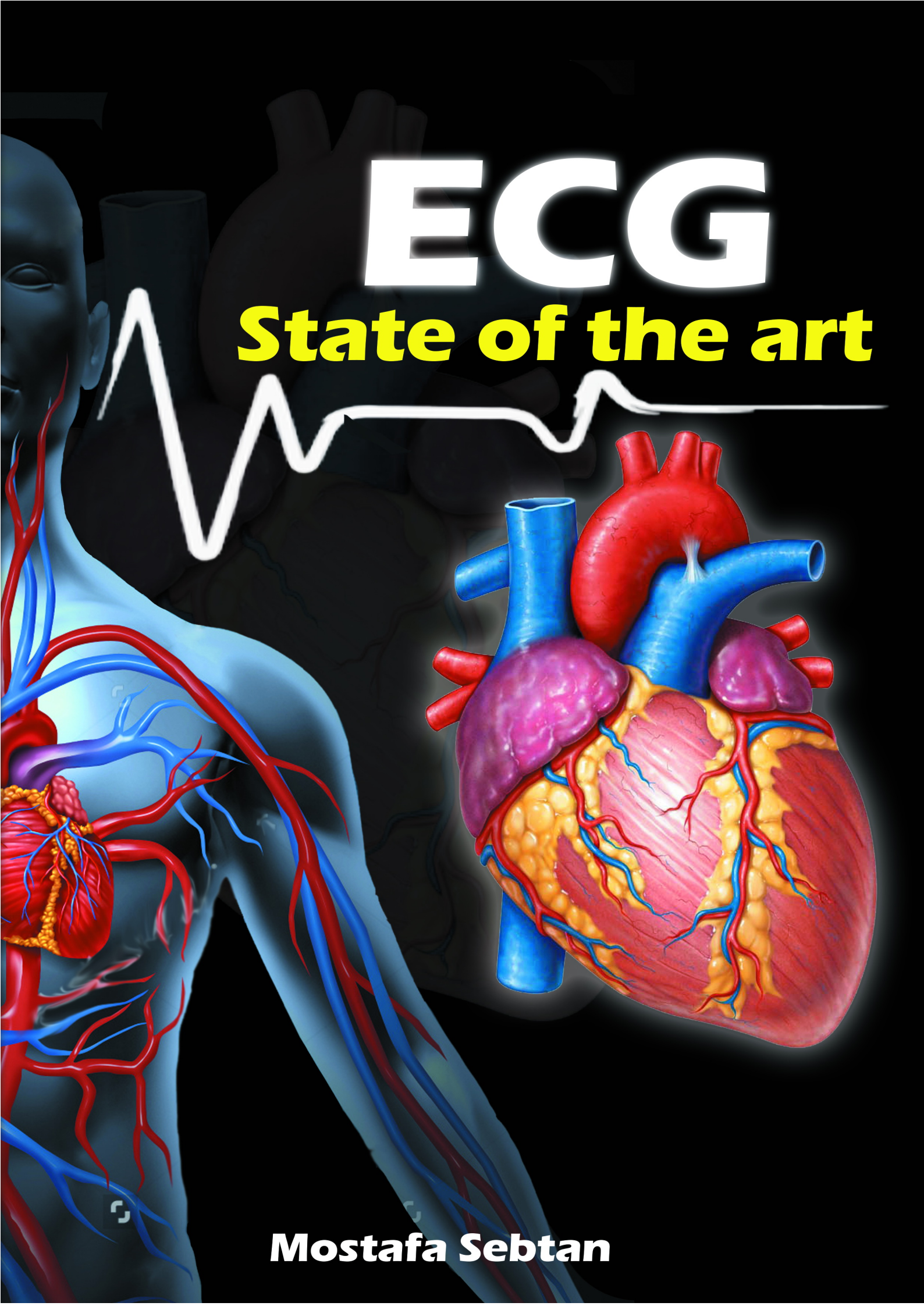
ECG

State of the art



Mostafa Sebtan

Preface

If you are a cardiologist, then ECG is a cornerstone in your career. If you are an undergraduate student or general practitioner or anyone else, then you will encounter ECG many times in the emergency states. It is essential to understand and learn how to interpret ECG abnormalities.

Many clinicians complain of learning ECG, understanding the mechanisms behind ECG changes which would result in wrong interpretation.

This book is designed to give you all you need about basics of ECG, diagnostic criteria of a variety of diseases encountered and the underlying electrophysiology behind such changes in a simple concise way. All of which are provided with many illustrating images and ECG strips.

I would like to express my deep gratitude to my brother Dr. Sherif Refaat lecturer of medical oncology, Oncology Center, Mansoura University for his huge support in reviewing, edition of this book and for his ultimate encouragement.

Words are powerless to express my gratitude to my dear professor Shahera El-Etreby Professor of Hepatology & Gastroenterology, Faculty of Medicine, Mansoura University for her guidance in editing this book and her precious endless support all time. Words can neither qualify nor quantify your guidance and useful advice.

Also many thanks and great appreciation to Dr. Wafaa Laimon Lecturer of pediatrics, Faculty of Medicine, Mansoura University for her fruitful contribution in reviewing this book. Your generosity overwhelms me.

Finally, you cannot construct a building without a good base. It is necessary to read all the introduction chapter before proceeding to ECG disorders. You may feel bothered while reading it for the first time and thinking about what is the benefit from such info. Just remember what I mentioned at the beginning of this paragraph. No base, no building.

Dedication

In character, in manner, in style, in all things, the supreme excellence is simplicity.

Henry Wadsworth Longfellow

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Chapter 1: Basic concepts of ECG

Contents

1) Basic concepts of the work of ECG machine.

ü How is electrocardiograph drawn? The answer is that waves with specific type and direction are converted into a diagram as follow: -

Ø Depolarization wave moving towards a lead gives rise to **positive deflection**.

Ø Repolarization wave moving towards a lead gives rise to **negative deflection** *(vice versa).*

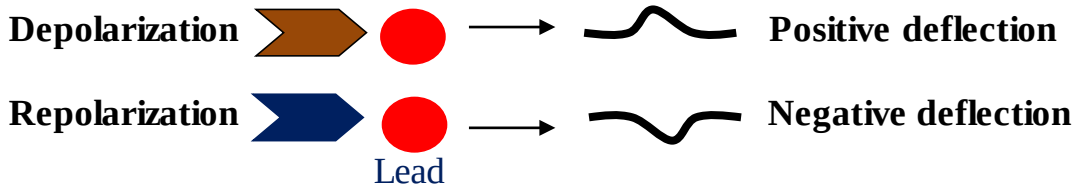


Figure 1-1: Concepts of the work of ECG machine

ü The waves spreads throughout the heart as follow **(Figure 1-2)**: -

1. **SAN discharges** impulse, spreads through the atria forming P wave (P wave travels in a downward direction).
2. **AVN receives** that impulse and conducts it to Hiss-Purkinji system giving the QRS complex via 3 stages:

Ø Septal depolarization occurs in interventricular septum from left to right bundle in a downward to upward direction.

Ø Major wave of depolarization spreads from the inner layer endocardium to the outer layer epicardium.

Ø Base of the heart is the last area to be depolarized

3. **Repolarization** occurs from epicardium to endocardium (opposite to depolarization direction) giving rise to T-wave.

ü By applying these principles to chest leads V1 located on right side and V6 located on left side **(Figure 1-2)** the result is:

1- P wave is heading downwards towards both leads so it is a positive wave.

2- Septal wave is heading towards V1 giving small +ve deflection (r wave), and away from V6 simultaneously giving small -ve deflection (q wave)

3- Major wave of ventricular depolarization (of dominant left side) is heading away from V1 giving 2nd large -ve deflection (S wave) and towards V6 simultaneously giving 2nd +ve deflection (R wave).

4- Repolarization wave spreads towards V1 giving -ve deflection (inverted T) and away from V6 simultaneously giving +ve deflection (upright T wave).

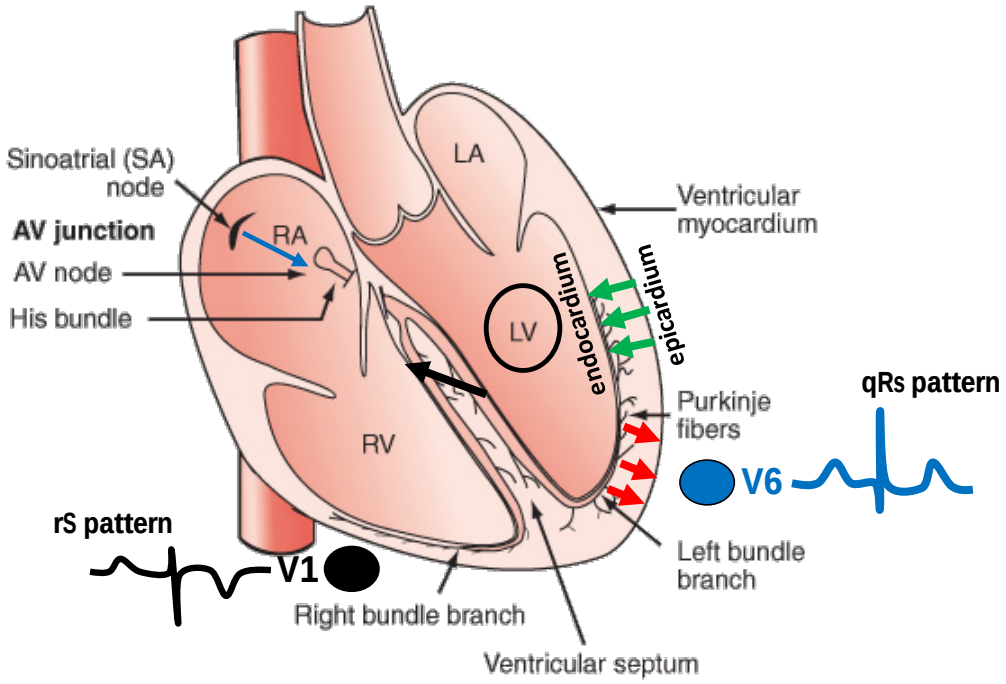


Figure 1-2: Normal pathway for impulse conduction

Red arrows represent direction of spread of depolarization waves (from endocardium to epicardium), **green arrows** represent direction of spread of repolarization waves, **blue arrow** represents atrial depolarization & **black** for septal depolarization. Left ventricular forces are the dominant (**black circle**). Quoted from *Fauci AS et al. Harrison's Principles of Internal Medicine, 17th ed. McGraw-Hill, 2008*

2) ECG leads

ü ECG leads are classified into two groups based on directions of waves moving inside the heart:

§ **Chest leads (V1 to V6)** cover transverse axis of the heart **(figure 1-3).**

§ **Limb leads** cover coronal (vertical) axis of the heart which are two subtypes

Ø Bipolar leads (L1 L2 L3) start from negative pole towards positive pole **(figure 1-4).**

Ø Unipolar (augmented) leads (aVF, aVL, aVR) at each limb

ü Location of these leads as follow

Chest leads (figure 1-3)

V1: right 4th intercostal space at sternal margin
V2: left 4th intercostal space at sternal margin
V3: mid-way between V2 and V4
V4: left 5th intercostal space at mid-clavicular line
V5: left 5th intercostal space at anterior axillary line
V6: left 5th intercostal space at mid-axillary line
They are unipolar leads with positive electrode on chest wall.

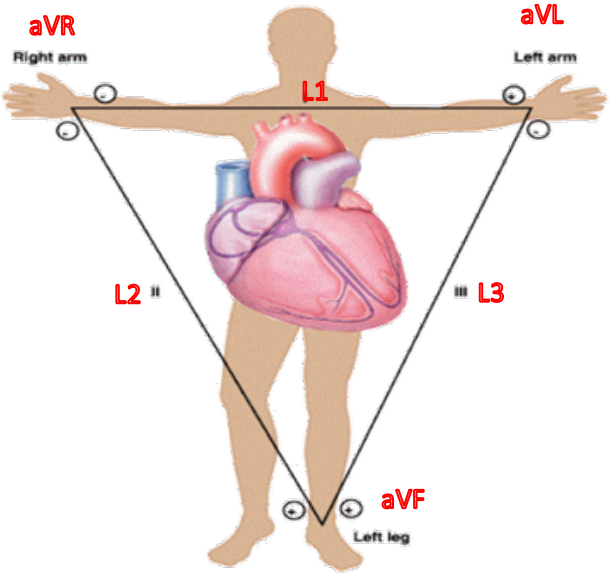


Figure 1-4: Limb leads
Quoted from *paramedicine101.blogspot.com*

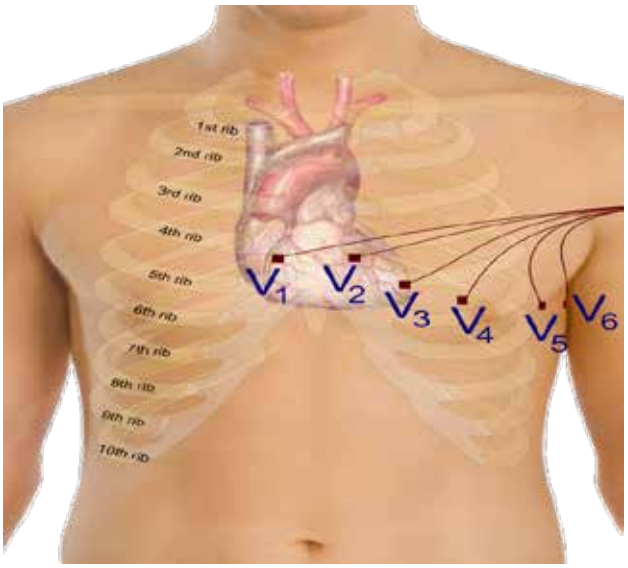


Figure 1-3: Chest leads
Quoted from *www.osceskills.com*

Limb leads (figure 1-4)

They are categorized into 2 groups:

1- Bipolar leads extending from limb to another. The first electrode is the negative while second one is the positive. They are:
   - **L1:** from right arm (-ve pole) to left arm (+ve pole).
   - **L2:** from right arm (-ve pole) to left leg (+ve pole).
   - **L3:** from left arm (-ve pole) to left leg (+ve pole).

2- Unipolar leads in which the positive electrode is located on the limbs.
   - **aVL:** located on left arm
   - **aVR:** located on right arm
   - **aVF:** located on left leg

ü Each lead gives a good idea about the electrical activity of a specific wall in the heart
   - ž L2, L3, aVF visualize inferior wall
   - ž L1, aVL visualize high lateral wall
   - ž V5, V6 visualize low lateral wall
   - ž V1, V2 visualize antero-septal wall (they are right sided leads).
   - ž V3, V3 visualize anterior wall of LV

3) ECG paper

Each big square has vertical line which represents amplitude and transverse line which represents duration. Every big square equals 0.5 mV and 0.2 sec. Every big square is divided into 5 small squares
(Each small square is 1x1 mm, 0.04 sec and 0.1 mV).

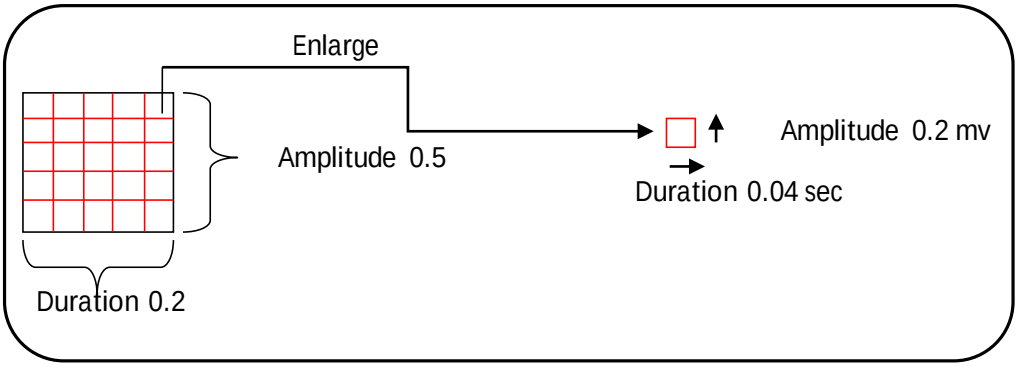


Figure 1-5: Calibration of ECG paper

4) ECG composition

ü **Waves, intervals and Segments (figure 1-6)**

Waves

- Ø **P wave:** 2.5 x 2.5 mm represents atrial depolarization
- Ø **QRS complex:** < 2.5 mm width represents ventricular depolarization
- Ø **q wave:** 1 x 1 mm represents septal depolarization.
- Ø **T wave:** 5 mm height in chest leads, 10 mm height in limb leads represents ventricular repolarization.
- Ø **U wave:** up to 3 mm height represents repolarization of papillary muscle.
- Ø **J point:** the point between the end of complex and the start of ST segment.

Intervals (composed of segment and wave)

- Ø **PR interval:** 3-5 mm width reflects normal AVN delay.
- Ø **QT interval:** up to 11 mm width indicator for ventricular repolarization.

Segments (isoelectric flat line)

- Ø **ST segment:** coincides with ventricular systole.
- Ø **PR segment:** represents ECG baseline (coincides with atrial systole).
- Ø **TP segment:** main ECG baseline (coincides with resting phase)

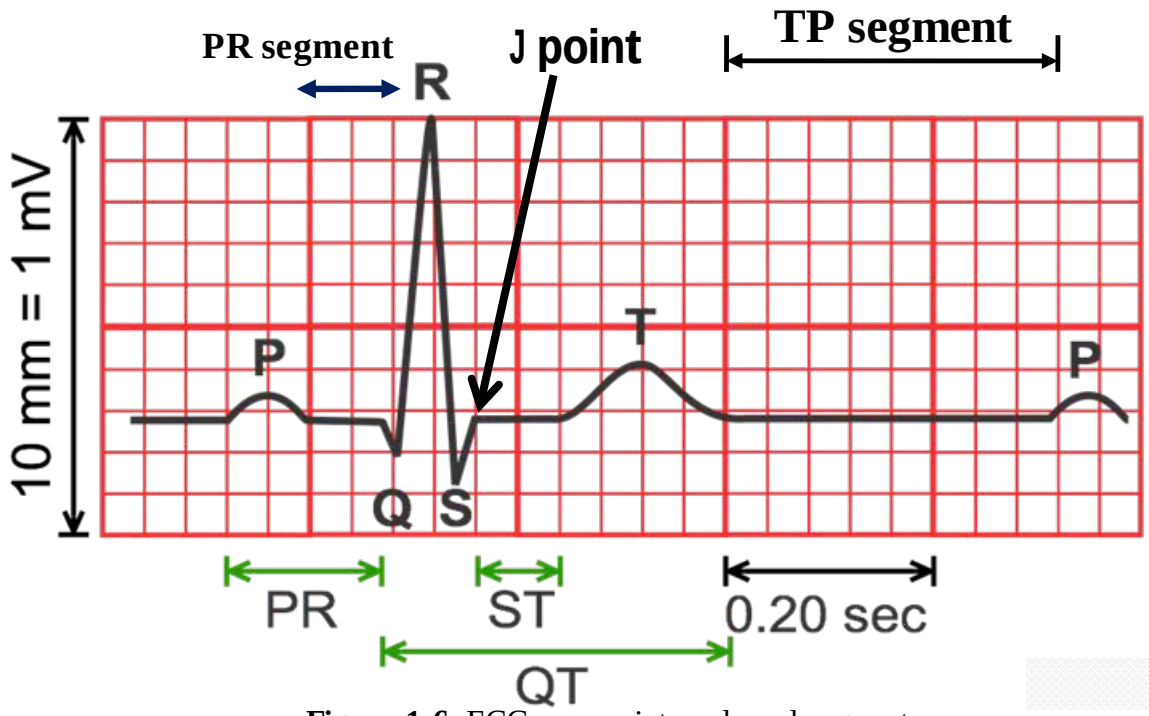


Figure 1-6: ECG waves, intervals and segments.
Quoted from *www.cvphysiology.com/Arrhythmias/A009.htm*

ü **Naming of QRS complex**

- First positive deflection = r wave, 2nd wave following it is S wave **(as in V1).** 3rd positive deflection may be abnormally found called r dash.
- First negative deflection = q wave, 2nd wave following it is R wave and 3rd wave would be s wave **(as in V6).**
- Only positive deflection = R wave
- Only negative deflection = QS wave
- QS, rS or qR complexes are biphasic complexes while QRS is triphasic.

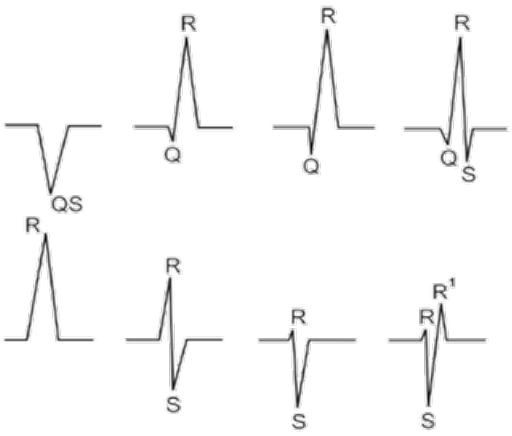


Figure 1-7: Patterns of QRS waves
Quoted from *Sajjan M, Learn ECG in a day*

ü **Normal ECG waveforms**

1- Calibration box (10 x 5 mm) to insure that device calibration is normal (recording speed equals 25mm/sec and each 10 mm height equals 1mV).

2- Be sure that aVR waves are inverted (as aVR is totally opposite to mean cardiac axis).

3- Analyze rhythm first before calculating rate from rhythm strip.

4- Q wave normally could be seen at V5, V6 and inferior leads.

5- Inspect neighboring leads for any abnormality (similar colors below).

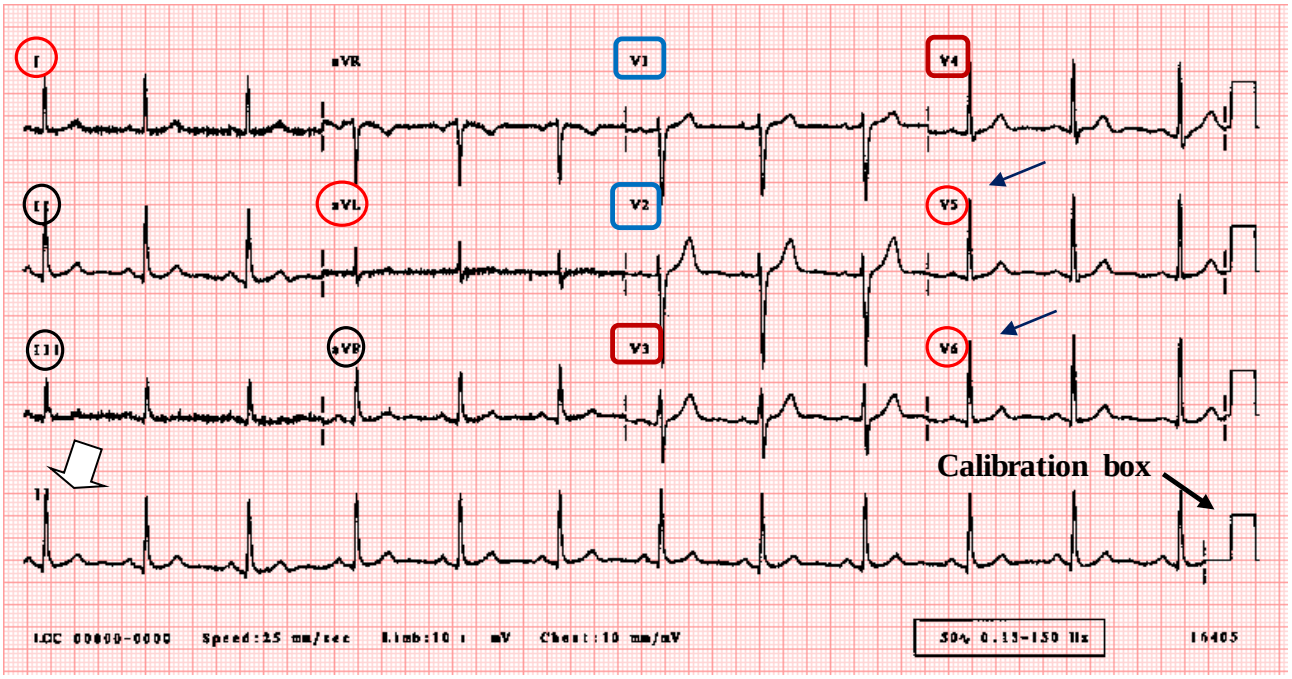


Figure 1-8: Normal 12 lead ECG configuration

Dark blue arrows in V5 and V6: low lateral wall. LII long tracing is called rhythm strip (white thick arrow)

Quoted from *www.ecglibrary.com*

5) What is vector?

Vector describes the direction and amplitude of any wave.

Ø If the depolarization wave vector is heading **towards** lead vector, then **a strongest positive deflection** will be drawn. (**Figure 1-9a).**

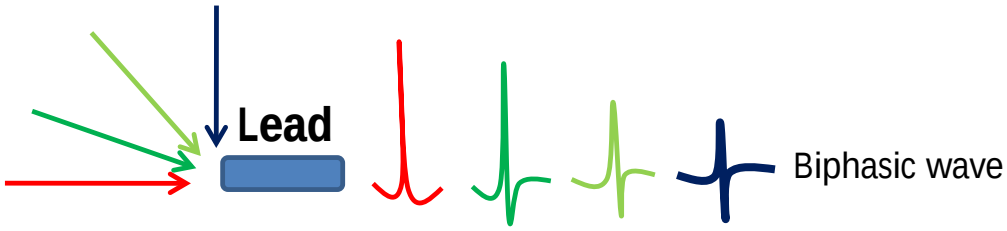


Figure 1-9a: Principles of vector

Ø If the depolarization wave vector is heading **perpendicular** on lead vector, this results in **biphasic wave** **(Figure 1-9b)**. The reason for it is that the proximal half of wave vector will be heading towards lead (thus +ve part) then the distal half will be heading away from lead (giving the -ve part)

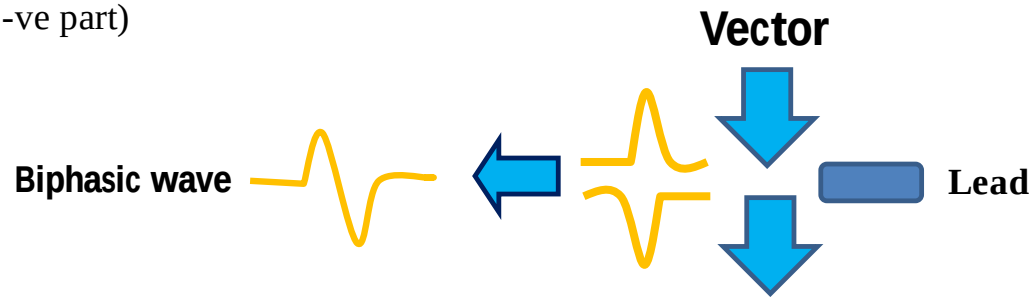


Figure 1-9b: Biphasic waves

6) Rhythm

Criteria of regular rhythm
1- Constant RR interval

Criteria of sinus rhythm
1- Each P wave is followed by complex
2- P wave are uniform in shape, duration and amplitude
3- Constant normal PR interval

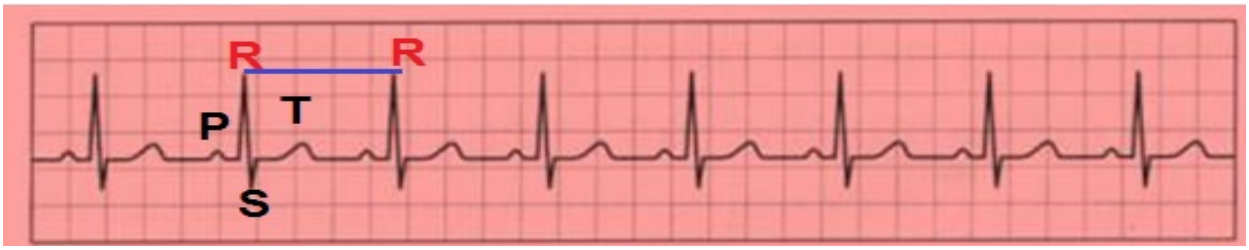


Figure 1-10a: Normal regular sinus

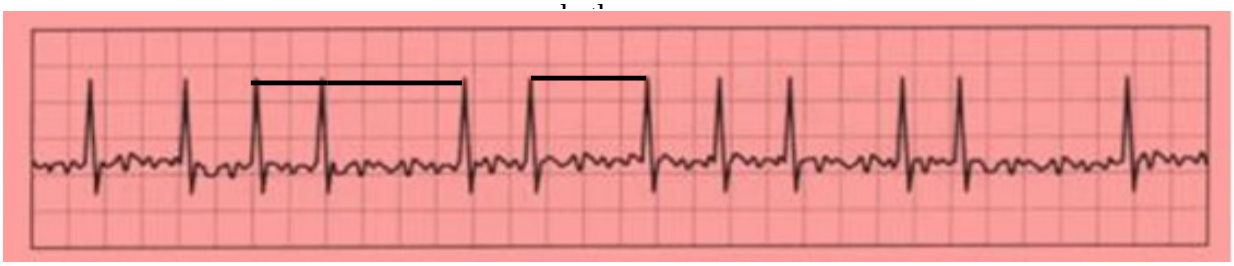


Figure 1-10b: Irregular rhythm (Atrial fibrillation)
Quoted from: *Sajjan M, Learn ECG in a day*

7) Rate

The simplest method is: (Number of R waves in 30 big squares x 10)
This method could be used roughly in regular and irregular rhythm.

Examples: -

In Figure 1-10a: There are 8 R waves in 30 LS so (8 x 10 = 80 BPM)
In Figure 1-10b: R waves are 12 so heart rate = (12 x 10 = 120 BPM)

Another method is: (300/ Number of big squares between RR). This method is applied in regular rhythm.

In figure 1-10a: 300 / 4 (no. of squares between RR) = 75 BPM

8) Axis

ü Recognition of axis deviation using 4 quadrant method.

1- Just look for QRS complex in L1 and aVF (or lead III).
2- Identify which complex has positive or negative dominance.
3- Plot these charges on the hexagonal diagram and identify normal and abnormal axis **(figure 1-11)**.

Normal axis ... -30 to +100 degrees **(between dotted red lines)**
Left axis deviation ...-30 to -90 degrees **(Upper right quadrant)**
Right axis deviation ...+100 to +180 degrees **(Lower left quadrant)**
Extreme axis deviation... -90 to +180 degrees **(Upper left quadrant)**

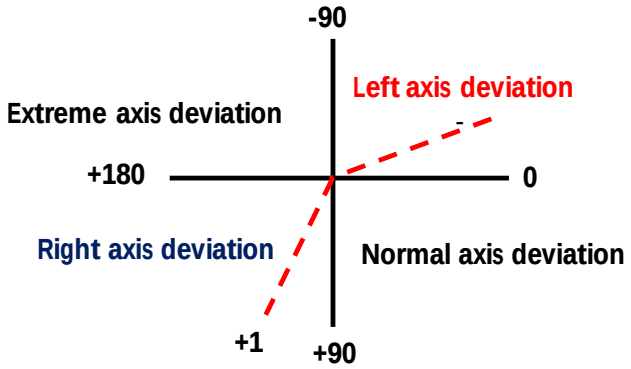


Figure 1-11: Four quadrant method of axis recognition

ü Factors affecting electrical axis

1- Anatomical position of the heart: Heart takes vertical position slightly with inspiration (due to diaphragmatic descent) shifting axis to the right, thus emphysema causes right axis deviation **(Figure 1-12).**

2- Direction of depolarization: delayed depolarization in specific area causes axis **deviation to that direction**, so right BBB causes right axis deviation.

3- Loss of vectors: shifting of axis to **opposite direction** so lateral wall MI causes right axis deviation.

4- Gaining of vectors: shifting of axis to the **same direction** of gain. Example for that is LVH in which more impulses goes to the left larger than normal ventricle so left axis deviation develops.

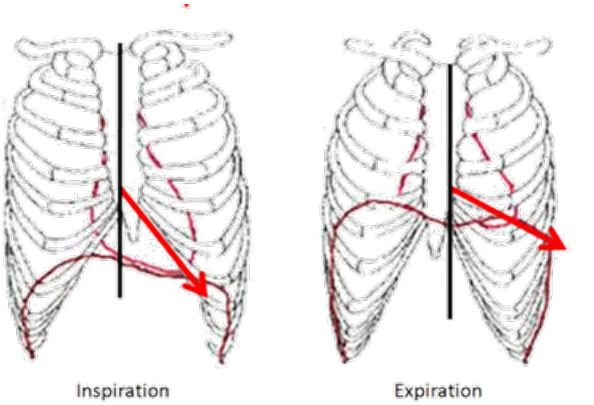


Figure 1-12: Role of anatomical position in axis deviation

Normally cardiac apex is shifted to the left (expiration figure). With inspiration, the diaphragm descends downwards so apex is shifted to the right. This will be more pronounced in patients with emphysema as hyperinflation of lung would cause significant diaphragmatic descent.

Quoted from *www.healthymoves-pa.com/documents/Diaphragm_and_Heart.htm*

Table 1: Causes of axis deviation

	Gain of vectors	Loss of vectors	Anatomical change	Direction of delayed depolarization
LAD	LVH	Inferior wall MI	----	Left anterior hemiblock Left BBB
RAD	RVH, TOF	Lateral wall MI	Emphysema	Right BBB Left posterior hemiblock

Abbreviations

BBB: Bundle branch block **TOF:** Tetralogy of fallot

RVH: Right ventricular hypertrophy **LVH:** Left ventricular hypertrophy

HOCM: Hypertrophic obstructive cardiomyopathy

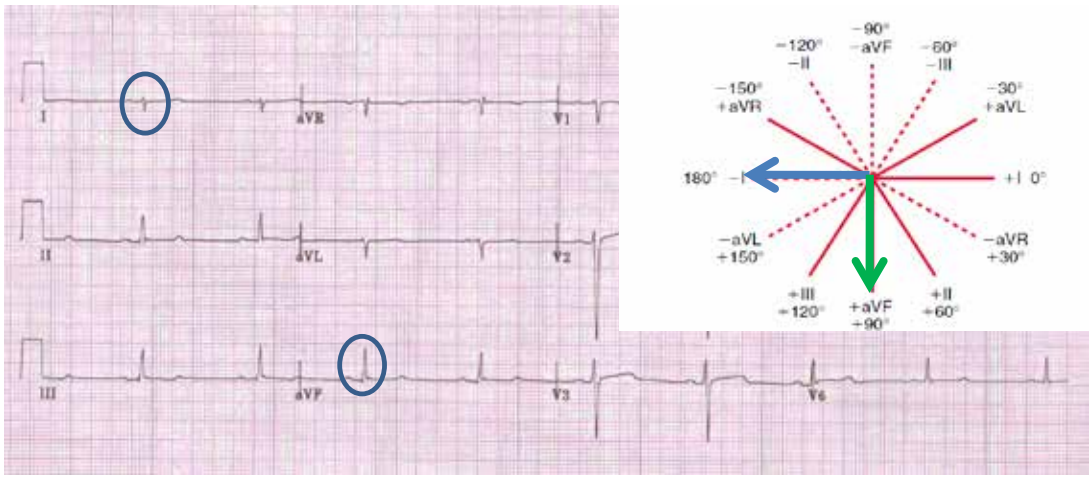


Figure 1-13: Right axis deviation

Complex is positive at aVF (green arrow) and negative at LI (blue arrow) so right axis deviation

12 lead ECG strip quoted from *lifeinthefastlane.com*

Illustrative image quoted from *Ary L. Goldberger's et al., Goldberger's clinical electrocardiography*

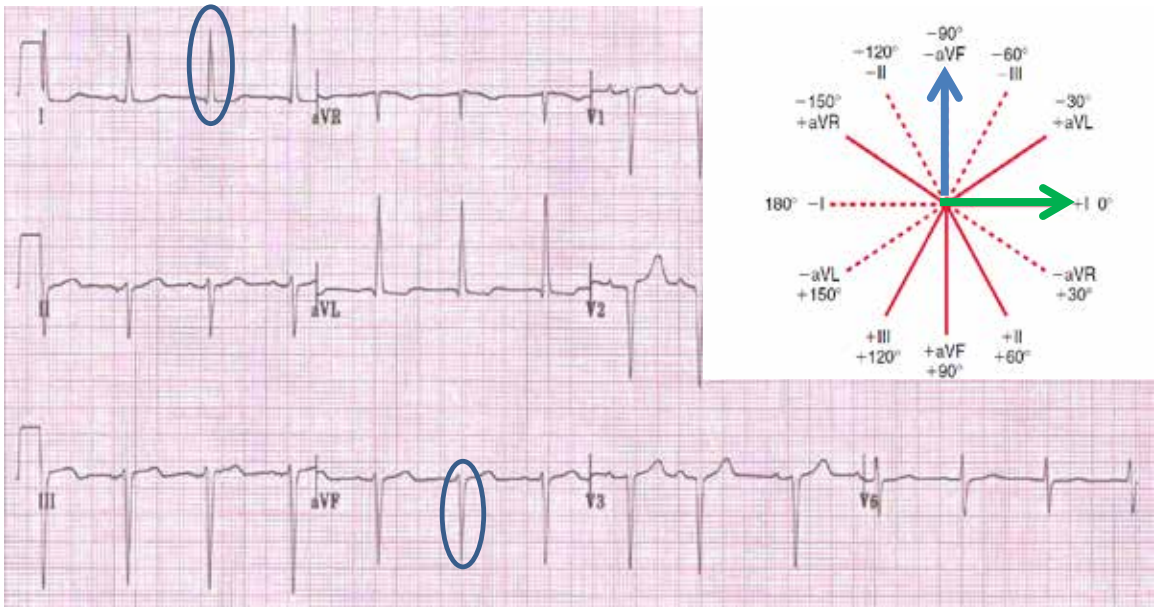


Figure 1-14: Left axis deviation

QRS complex is positive at LI (green arrow) and negative at aVF (blue arrow) so left axis deviation

12 lead ECG strip quoted from *lifeinthefastlane.com*

Illustrative image quoted from *Ary L. Goldberger's et al., Goldberger's clinical electrocardiography*

9) Stepwise approach to ECG paper

- Ø Look at calibration box and aVR waves should all be inverted
- Ø Rhythm analysis
- Ø Rate
- Ø P wave
- Ø PR interval
- Ø QRS complex
- Ø T-wave & ST segment and QT interval.

Clinical pearls

1- Normal ECG does not exclude heart disease.

2- U wave may appear normally during bradycardia and abnormally as in hypokalemia

3- Respiratory sinus arrhythmia is a normal physiological condition in which the rhythm is irregular but still of sinus origin. It occurs as a result of influence of respiratory cycle on heart rate (heart rate increases with inspiration and decreases with expiration).

4- Axis deviation to the right or left is not always a sign of abnormal pathology.

5- Sometimes all six limb leads show biphasic complex (QR or RS) so axis cannot be assessed. This finding is called indeterminate axis.

Chapter 2: P wave

Contents

1) P-wave

- Parameters = **up to 2.5 x 2.5 mm**. (height x width).
- Represents atrial depolarization.
- Biphasic at V1, Negative at aVR & Positive at rest of leads.
- First part formed by right atrium while second by left atrium.

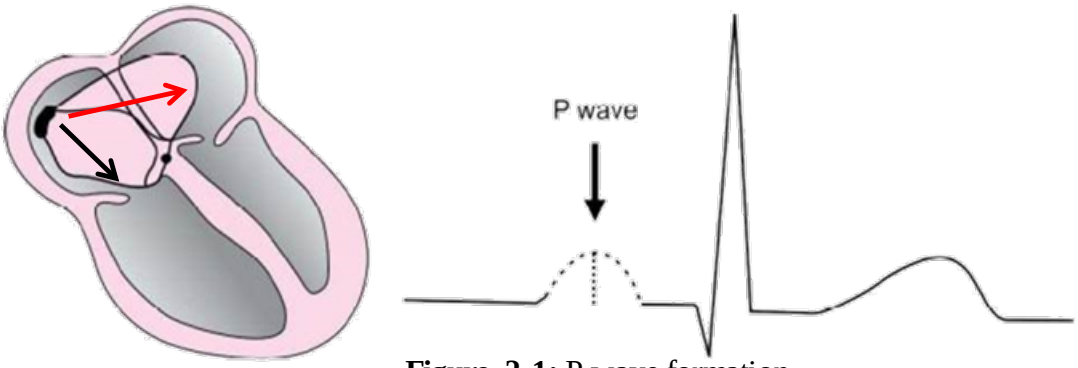


Figure 2-1: P wave formation

Wave spreads in right atrium (down & right as black arrow) forming 1st +ve deflection then in left atrium (down and left as red arrow) forming 2nd –ve deflection at V1

Quoted from *Sajjan M, Learn ECG in a day*

2) Atrial enlargement

Table 2: Characteristic features of right and left atrial enlargement

Right atrial enlargement	Left atrial enlargement
Increase in P wave **height** **V1:** +ve phase > **1.5 mm** **Inferior leads:** P > **2.5 mm**	Increase in P wave **width** mainly or with duration. **V1:** -ve phase > **1x1 mm** **Inferior leads:** P wave > **3 mm** width

Electrophysiology

SAN is located in right atrium so impulse reaches it first, so RAE is usually expressed as increased amplitude (Tall P wave). After that, the impulse reaches left atrium via Bachmann's bundle so takes time to travel to it so any enlargement in left atrium would affect duration mainly (Wide P wave).

NB

1- One feature is enough to diagnose atrial enlargement
2- Presence of features of both often denotes bi-atrial enlargement
3- Massive right atrial enlargement may produce inverted P wave in V1

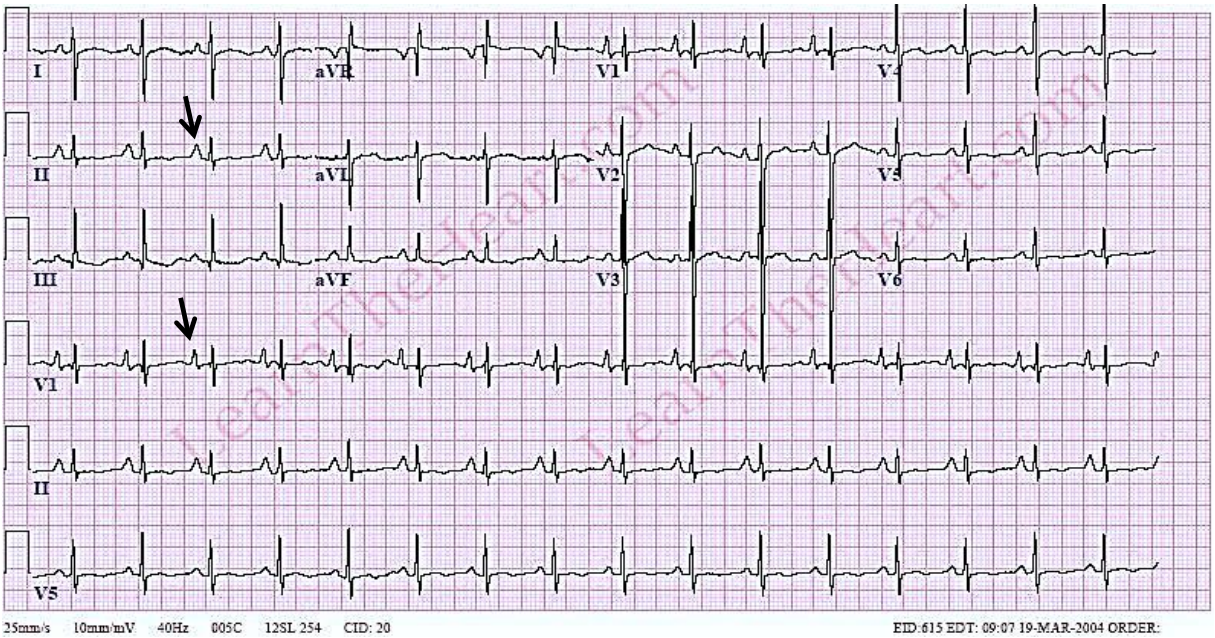


Figure 2-2: Right atrial enlargement

Tall P wave>2.5 mm in inferior leads + tall +ve phase at V1 >1.5 mm

Quoted from *lifeinthefastlane.com*

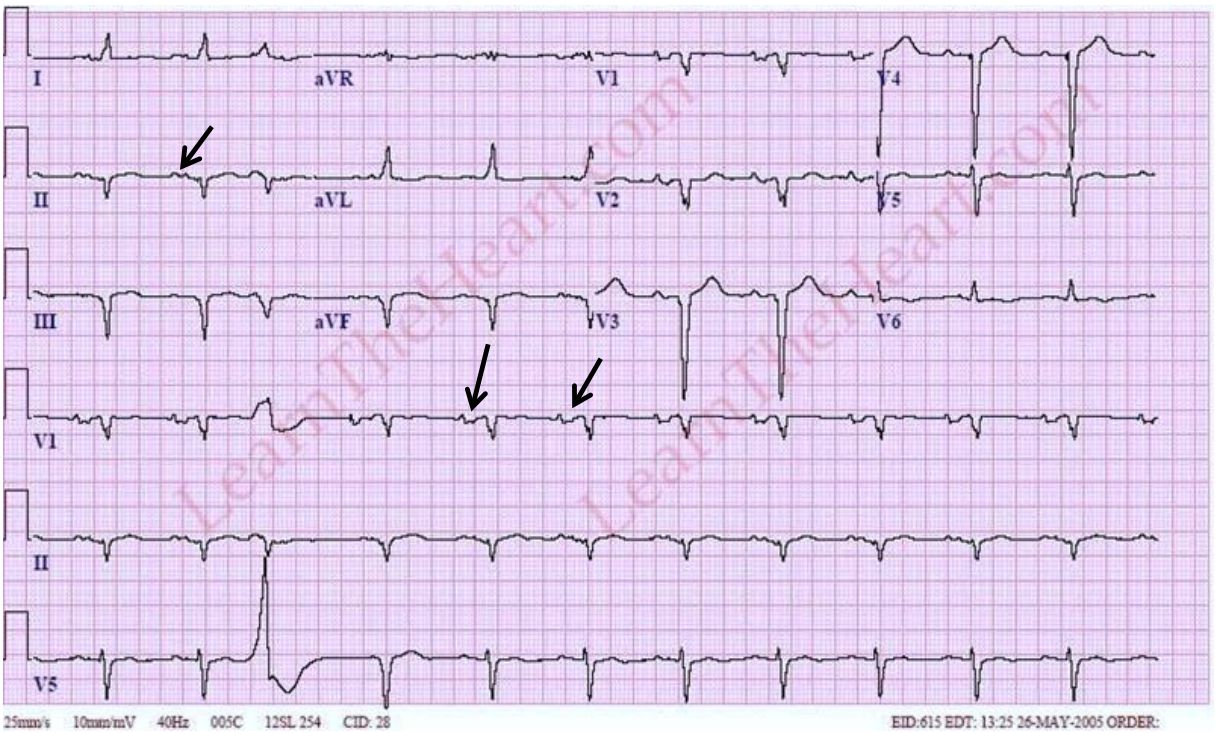


Figure 2-3: Left atrial enlargement

Wide notched P wave <3mm at LII + wide deep negative phase >1x1 mm at V1

Quoted from *lifeinthefastlane.com*

When the right atrium gets too large, it grows to the left side (red arrow), so the axis of the wave spreading throughout the right atrium is shifted to the left. This makes P wave totally shifted to the left (both of right & left atria) away from V1 (which is on the right side of chest wall) giving fully negative P wave at V1 instead of biphasic P wave (Figure 2-4).

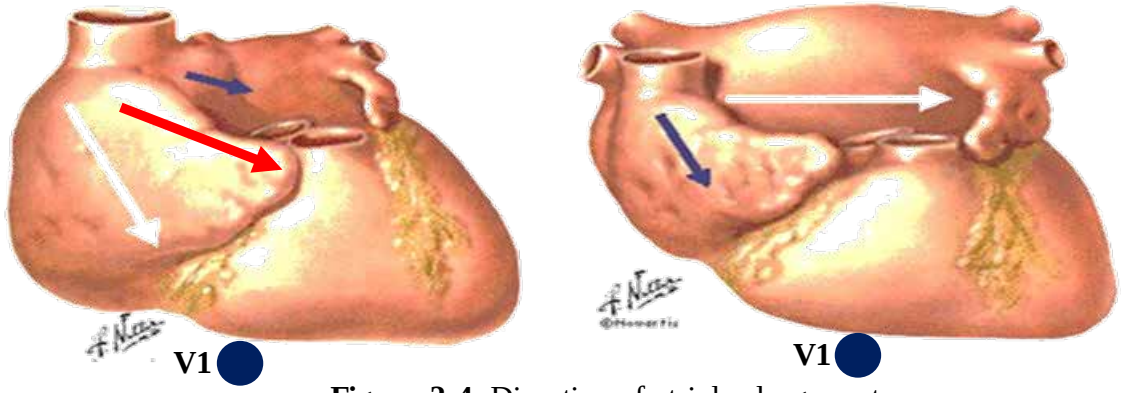


Figure 2-4: Direction of atrial enlargement

Right image represents normal right atrium, the left represents enlarged right atrium (red arrow pointes to direction of right atrial enlargement to the left).

Quoted from *Frank Netter,The Netter Collection of Medical Illustrations - Heart*

Clinical pearls

1- Mitral stenosis and ventricular septal defect are causes of left atrial enlargement.
2- Tricuspid stenosis is a cause of right atrial enlargement.

Chapter 3: PR interval

Contents:

1) PR interval

- ü Definition: the duration between the start of P wave to the start of complex.
- ü Parameters = 0.12-0.2 sec **(3 – 5mm).**

It represents the duration taken for 4 events to happen: -

1) Generation of SAN impulse
2) Atrial depolarization
3) AVN delay
4) Propagation of impulse to AV bundle (bundle of Hiss), bundle branches and Purkinji fibers.

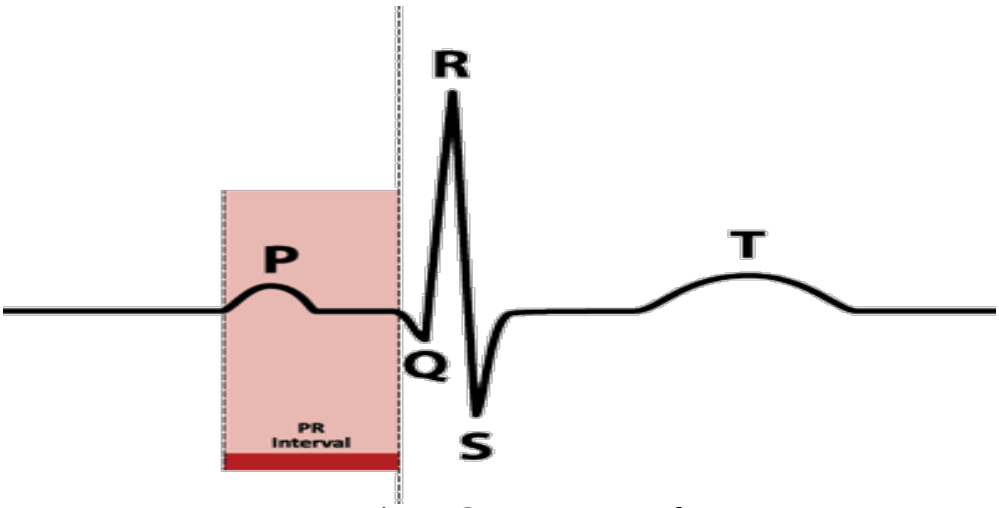


Figure 3-1: - PR interval
PR interval is marked by pink area
Quoted from *ekg.academy/ekg-waveform-lesson*

- Ø **Short PR interval** means that ventricles are depolarized earlier than expected (Pre-excitation syndromes)
- Ø **Long PR** interval means that ventricles are depolarized later than expected (AVN block).

2) AVN block

3 degrees are known for AVN block

Ø First degree

Definition: only prolonged constant PR interval >0.2 sec **(>5mm)**

Occurs with any condition slowing AVN conduction as vagal stimulation.

Ø Second degree

Definition: **Some** atrial impulses **fail** to reach ventricles.

2 subtypes:

Mobitz I	Mobitz II
ü **Progressive PR prolongation** till non-conducted P occur (Classic Wenckebach phenomenon)	ü **PR interval remains unchanged** prior to a non-conducted P wave
ü Variable PR interval.	ü Constant PR interval.
ü The affected site usually is AVN	ü The affected site is infra nodal

Ø Third degree

Definition: **No** atrial impulses reach ventricles (complete dissociation between atria and ventricles)

ECG features:

1. No fixed relation between P and complexes.
2. Variable PR interval.
3. Ventricles work by escape rhythm instead of SAN (slow HR). rate).

N.B: - Any depolarization abnormality would result in 2ry ST segment and T wave changes so do not evaluate ST-T changes solely before evaluating any abnormal depolarization (such as SVT or PVCs).

Electrophysiology

Mobitz I: - Diseased AVN cells become more fatigued after each propagated atrial beat (progressive lengthening of recovery period) till they become totally unavailable for conduction. At this stage, a non-conducted atrial beat occurs.

Mobitz II: His-Purkinji cells (infra-nodal structures) are characterized by **On and Off phenomenon. If** they are On (recovered), impulse is conducted. If they are off (refractory to conduction), no propagation at all occurs). Diseased cells suddenly fail to propagate an impulse causing this type of block.

Complete heart block: - AVN does not conduct any impulse at all. Atria are depolarized via SAN while ventricles are depolarized via **escape rhythm Table 3**. Subsidiary pacemaker begins to fire impulses; it could be AVN (junctional escape rhythm) or ventricles itself (ventricular escape rhythm)

- As SAN firing is higher than AVN and any other pacemaker so P waves would be more in number than QRS complexes but no relationship between them (you can't expect when P wave will come before QRS complexes).

Escape rhythm: when the sinus impulse gets blocked from being propagated down to ventricles, subsidiary pacemakers begin to fire and gain control over ventricles. Examples of theses subsidiary pacemakers are AVN (junctional) or ventricular myocardium itself (ventricular).

Table 3: Escape rhythm

Junctional escape rhythm	Ventricular escape rhythm
Heart rate is 40-60 BPM.	Heart rate is 20-40 BPM.
QRS complexes are typically narrow (< 3 mm) No relationship between the QRS complexes and any preceding atrial activity (e.g. P-waves, flutter waves).	QRS complexes are broad (>3 mm) May have BBB morphology.

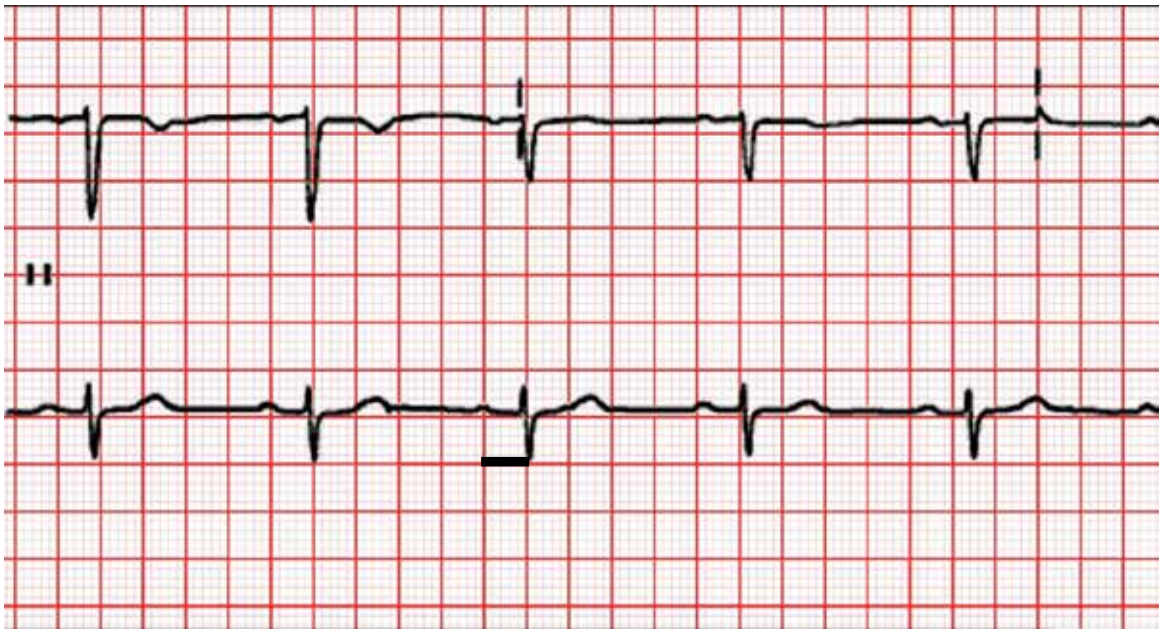


Figure 3-2: First degree heart block
PR interval is 0.24 sec (6mm) and constant
Quoted from *en.ecgpedia.org*

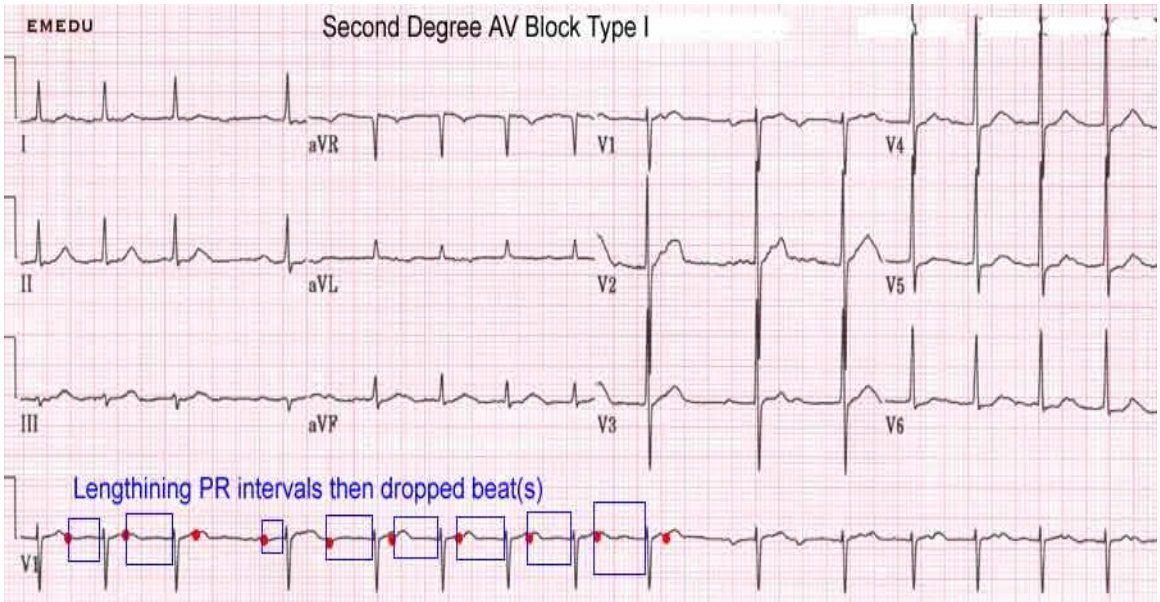


Figure 3-3: Mobitz I (2nd degree HB)
Progressive PR prolongation till dropped beat occurs (Wenckebach phenomenon)
Quoted from *www.emedu.org*

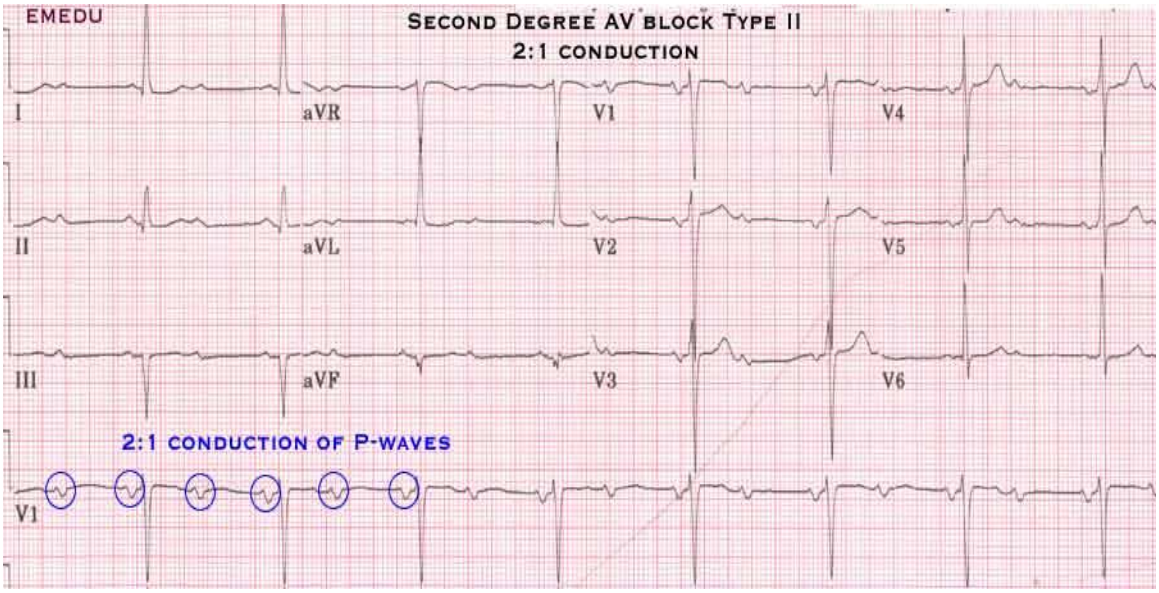


Figure 3-4: Mobitz II (2nd degree HB)
PR interval is constant before non-conducted atrial beats occur
Quoted from *www.emedu.ora*

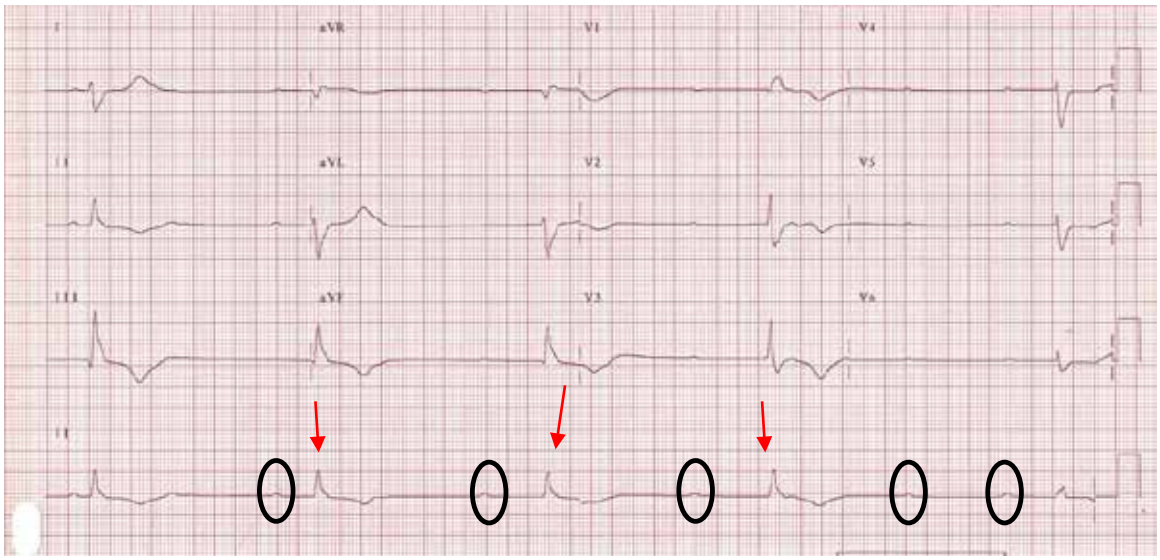


Figure 3-5: 3rd degree HB
Regular RR intervals. No fixed relation between P (circles) and complexes (arrows).
Also very slow rate 27 usually of ventricular origin (ventricular escape rhythm)
Quoted from *lifeinthefastlane.com*

3) Pre-excitation syndromes

Ø **Wolff Parkinson White syndrome**
Short PR interval + delta wave + wide complex + inverted T wave
Delta wave is slow slurring rise of initial portion of QRS complex

Ø **Lown-Ganong-Levin syndrome**
Only short PR interval + otherwise normal ECG

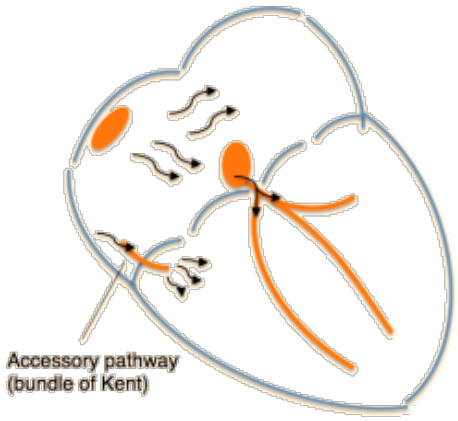


Figure 3-6: Pre-excitation pathways
Quoted from *www.ems12lead.com*

Electrophysiology

1- Accessory pathway connects atria to ventricles
2- In WPWS, bundle of Kent connecting atria to ventricle directly bypassing AVN which leads to:
   - Ø Short PR interval (due to AVN bypass)
   - Ø Delta wave (abnormal initial slurring in complex) due to initial abnormal ventricular activation via myocardium
   - Ø Wide Complex due to slow myocardial conduction
   - Ø Inverted T wave due to reversal of depolarization pathway
3- In Lown-Ganong-Levin syndrome, there is either fast AVN conduction or direct connections (atrial to lower AVN part or atrial to AV bundle). The result is fast ventricular activation through normal pathway so short PR interval, normal narrow QRS complex and T wave.

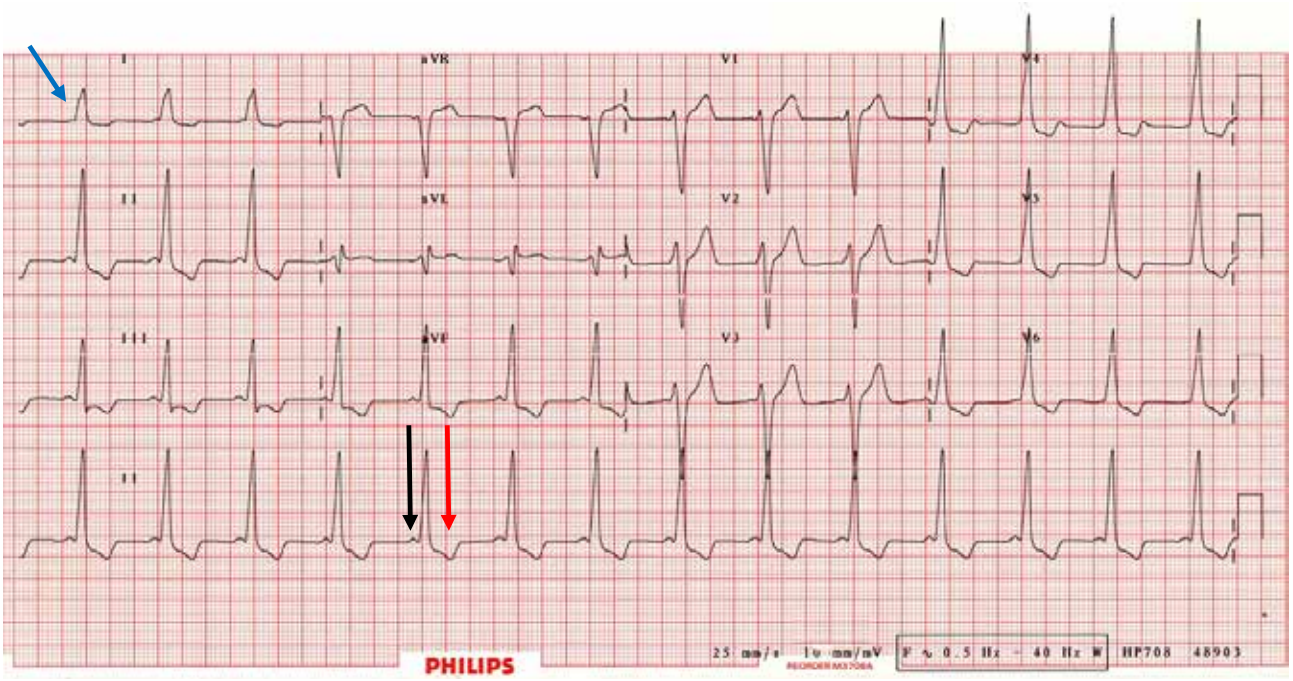


Figure 3-7: Wolff Parkinson White syndrome
Short PR interval (note black arrow on P wave) + delta wave (blue arrow) + inverted T (red arrow)
Quoted from *lifeinthefastlane.com*

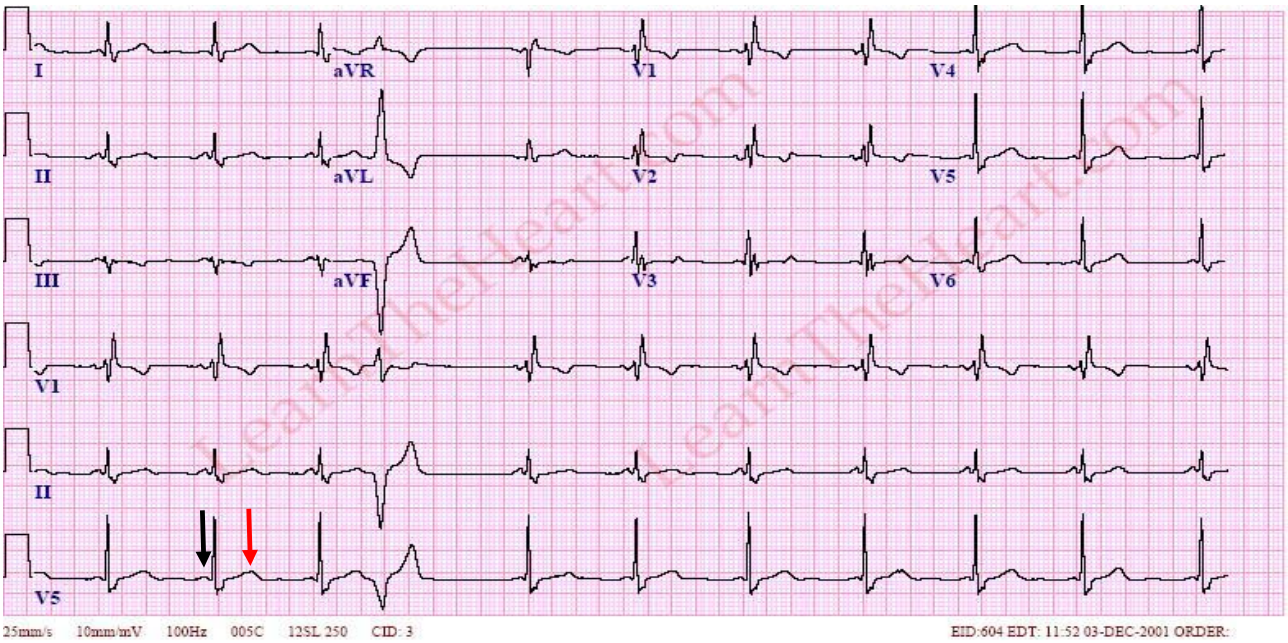


Figure 3-8: Lown-Ganong-Levin syndrome
Short PR interval (note black arrow on P wave) + upright T (red arrow)
The 4th beat with abnormal complex and T comes earlier than normal (VPC)
Ventricular premature beat
Quoted from *lifeinthefastlane.com*

Clinical pearls

1- Wolff Parkinson White syndrome is a cause of atrioventricular reentry tachycardia.
2- Atrial fibrillation in the presence of WPWS can result in very high ventricular rate enough to compromise cardiac output causing syncope or even death.
3- Mobitz II and complete heart block are at high risk of cardiac arrest and often necessitate permanent pacemaker.

Chapter 4: QRS complex

Contents:

1) QRS complex

- **Duration:** less than 3 mm (0.06 to 0.1secs).
- **Naming of QRS complex**
  - First positive deflection = r wave, 2nd wave following it is S wave **(as in V1)** and 3rd positive deflection wave abnormally would be R dash.
  - First negative deflection = q wave, 2nd wave following it is R wave and 3rd wave would be s wave **(as in V6).**
  - Only positive deflection = R wave
  - Only negative deflection = QS wave
  - Almost always 1st wave in complex is of septal depolarization & 2nd represents ventricular myocardial depolarization.

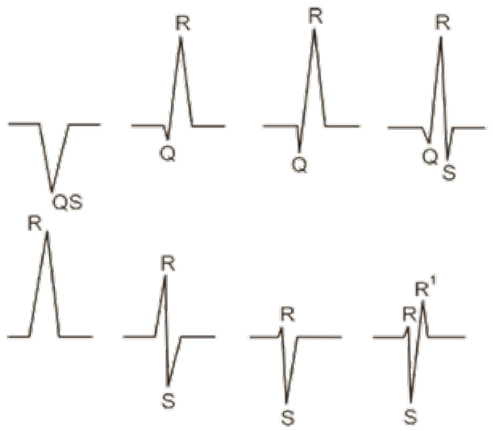


Figure 4-1: Different shapes of QRS complex
Quoted from *Sajjan M, Learn ECG in a day*

2) Blood supply of the heart

- **Origin:** Ascending aorta
- **Branches:**
  - **Right coronary artery**
    * It supplies inferior (diaphragmatic) portion of the heart and right ventricle mainly.
    * It gives **posterior descending branch** to posterior wall of the heart.
  - **Left main coronary gives off two major branches**
    * **Left anterior descending** for interventricular septum and anterior wall of left ventricle.
    * **Left circumflex** for lateral wall of left ventricle.

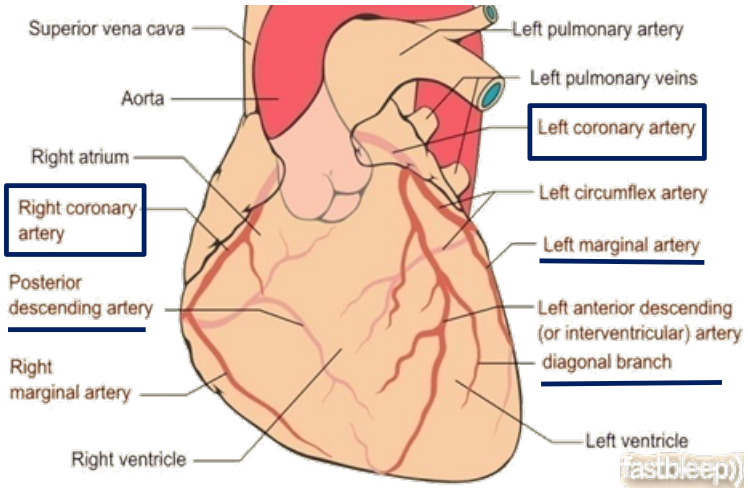


Figure 4-2: Coronary artery anatomy
Quoted from *fastbleep.com/biology-notes/1/572*

3) Myocardial infarction

ü **Definition:** Infarction means cutting of blood supply results in death of myocardial cells. It is different from ischemia in which cells are still viable.

ü **Classification**

STEMI (ST elevation (MI): - Complete coronary obstruction causes transmural (full thickness) infarction.

NSTEMI (Non ST elevation MI): - Partial coronary obstruction causes subendocardial infarction.

STEMI features

Early changes

1- Hyperacute T wave
- >5 mm in limb leads
- >10 mm in chest lead

2- ST elevation
- Starts at J point
- ≥ 1 mm in all leads
- ≥ 2 mm in V2 & V3

Late changes

1- Pathological Q wave
- >1x1 mm

2- T wave changes
- inverted, flat then normalizes

NSTEMI features

1-ST depression
- Measured after J point
- ≥0.05 mV

2-T wave inversion

N.B: changes should be at least in two contagious leads

Electrophysiology

1-Ischemia leads to shortening of action potential duration (AP duration), early accelerated repolarization and lower resting membrane potential (RMP) of these areas.

2-STEMI (transmural ischemia) Figure 4-3**a**:

- ü During systole (corresponds to ST segment), ischemic zones are more positively charged than non-ischemic (due to accelerated early repolarization) so **systolic** current of injury passes from negative non-ischemic to positive ischemic zones towards the lead producing **true ST elevation**.
- ü During diastole (corresponds to TP segment), non-ischemic zones are more positive relative to ischemic (due to lower RMP of ischemic areas) so producing **diastolic** current of injury from ischemic to non-ischemic away from lead depressing TP segment so **ST is apparently elevated**.

3- NSTEMI (subendocardial ischemia) Figure 4-3**b**:

- Ø Systolic current of injury passes from normal epicardium (negative charge) to ischemic endocardium (positive charge) producing **true ST depression**.
- Ø Diastolic current of injury passes from endocardium (which becomes negative relatively to epicardium) to epicardium causing TP elevation so **ST is apparently depressed**.

4- Necrotic (dead) zone is electrically inert so acts as a window from which any wave traversing ventricular cavity will be recorded. As depolarization wave is heading away from this zone, Q wave is drawn.

5- Ischemia increases K permeability producing tall T wave.

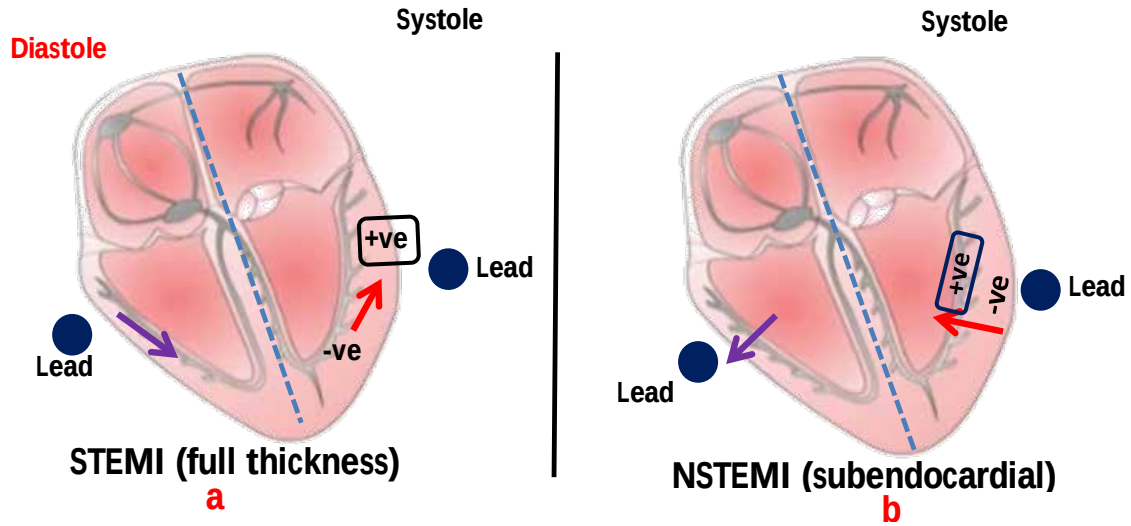


Figure 4-3: Current of injury in myocardial infarction

Highlighted areas represent ischemic regions which have short AP duration and repolarizes early.
Quoted from *ecgteachear.com*

ü **Reciprocal relation**

- Two electrodes facing MI zone from opposite sites. ST elevation and upright T wave in **a lead** will appear as ST depression & inverted T wave in the **opposite lead**.
- Anterior wall chest leads (V1 to V4) are located above the inferior leads (LII, LIII, aVF) so reciprocal relation exists between them.
- High lateral wall leads (L1 and aVL) are up & left opposite right sided chest leads (V1 & V2) and also inferior leads (LII, LIII and aVF).
- Posterior leads (V7- V9) are located opposite antero-septal leads (V1 and V2).

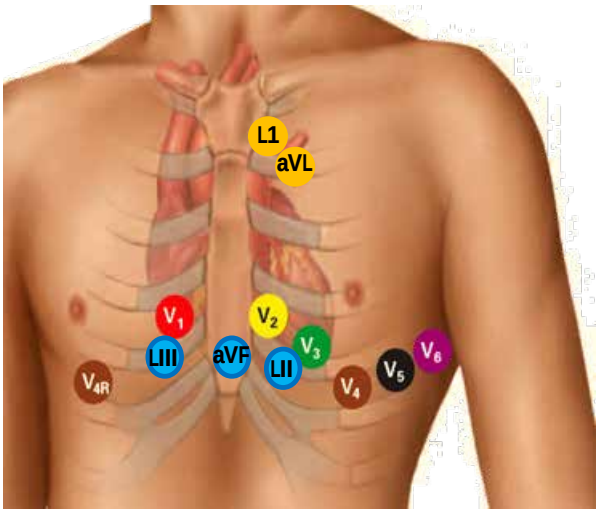


Figure 4-4: Reciprocal relation
Quoted from *wikis.engrade.com/ekgelectrodes*

How to suspect right ventricular infarction?

ü ST elevation in right leads (V1 and LIII). ST elevation in LIII >LII

ü ST elevation in V1 + ST depression in V2 is highly specific for RV MI (reciprocity due to RV affection and not septal wall MI)

ü As RCA supplies inferior wall and right ventricle, inferior wall MI should be followed by inspection for possible Right ventricular infarction (put right sided leads V3R and V4R to reveal ST elevation).

How to suspect posterior wall MI?

Posterior MI is suggested by the following changes in V1-3:

- ST depression **(mirror of ST elevation on posterior wall)**
- Tall, broad R waves (>30ms) **(mirror of Q wave)**
- Upright T waves **(mirror of inverted T wave)**
- Dominant R wave (R/S ratio > 1) in V2

Table 4: Localization of site of infarction (see also **figure 5-4).**

Site	Direct leads	Reciprocal leads	Affected artery
Anterior	V1 to V4	Inferior leads	LAD
Antero-septal	leads V1-V2	Inferior or lateral leads	LAD
Antero-apical	V3 and V4	Inferior leads	LCC LAD or DRCA
Antero-lateral	V5, V6, LI and aVL	Inferior leads + V1 &V2.	LCC LAD or DRCA
Lateral	leads I and aVL	Inferior leads + V1 &V2.	**LCC**
Inferior	II, III, and aVF	leads I & aVL	RCA or LCA
Posterior	leads V7-V9	V1-3	LCA

Abbreviations
LAD: left anterior descending – LCC: left circumflex coronary – RCA: Right coronary artery - DRCA: Dominant right coronary artery

N.B: **Myocardial ischemia** has the same changes of NSTEMI. The difference is that cardiac muscle is still viable so chest pain often disappears on sublingual nitroglycerine.

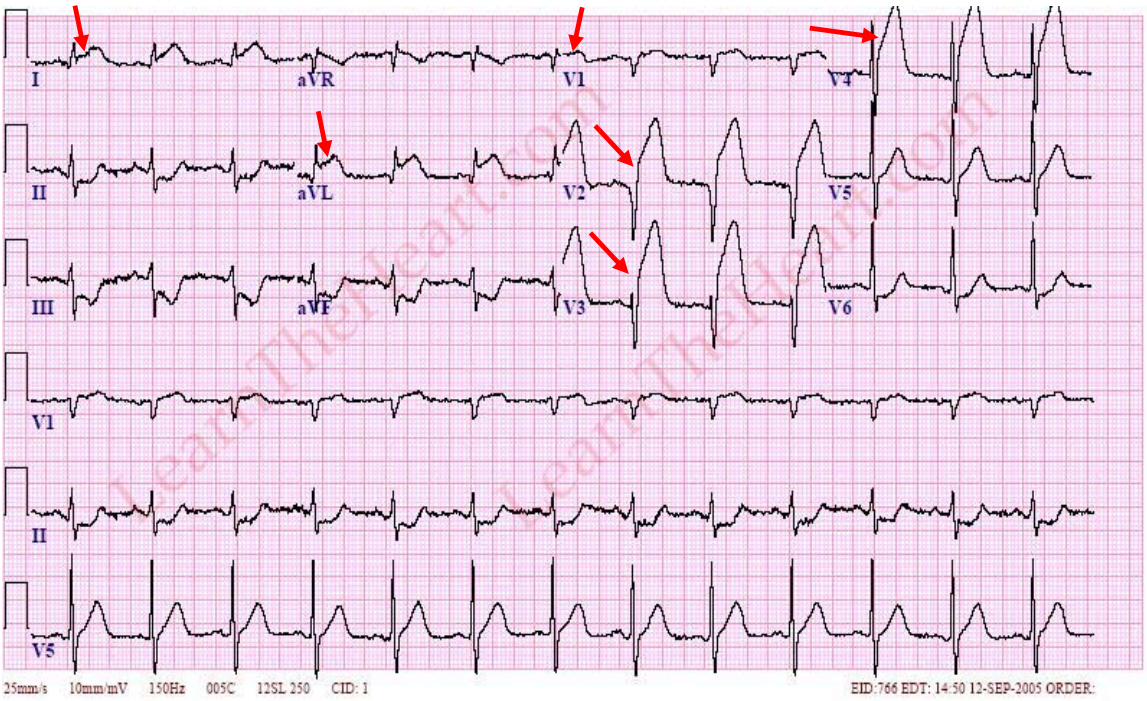


Figure 4-5: STEMI of anterior and high lateral walls
ST elevation is found in V1toV4, L1 and aVL leads (arrows), note reciprocal depression in inferior (II.III and aVF) leads
Quoted from *www.healio.com/cardiology*

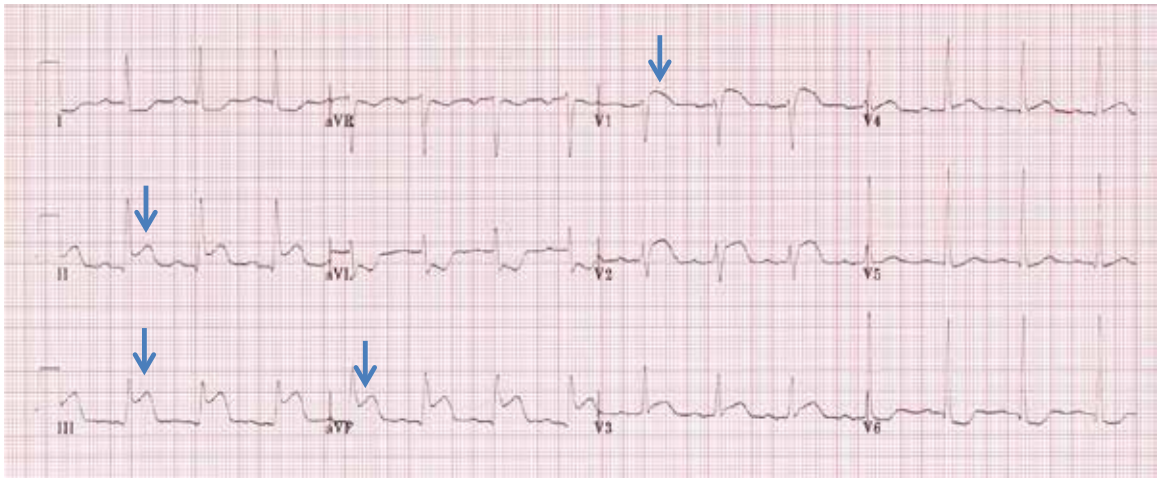


Figure 4-6: Right ventricular & inferior wall infarction
ST elevation is found in V1 and inferior leads + ST elevation in LIII >LII
Quoted from *lifeinthefastlane.com*

4) Bundle Branch Block

ü Anatomy

The Hiss bundle divides at the upper part of interventricular septum into left and right branches.

The left bundle is further divided into 3 fascicles: septal, anterior and posterior.

Anterior fascicle is located at upper lateral wall of LV.

Posterior fascicle is located at inferoseptal wall of LV.

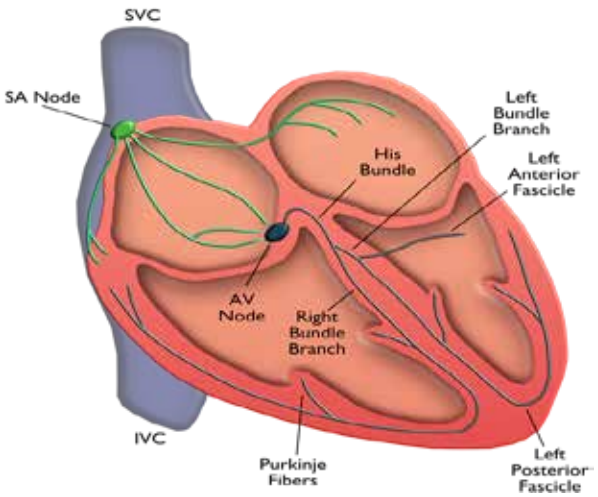


Figure 4-7: Anatomy of His-Purkinji system
Quoted from *mdnxs.com/topics-2/cardiology/cardiac-physiology/*

ü Types of bundle branch block

Table 5: Left versus Right bundle branch block

	Left BBB	Right BBB
QRS complex	Wide >0.12 sec	Wide >0.12 sec
Pattern QRS	Notched or slurred R in lateral leads QS or rS in V1 and V2	Notched R or rSR in V1 &V2 S wave duration >R duration in L1 & V6
ST segment	Depressed in V5 or V6	Little change (depressed)
T wave	Inverted in left leads	Inverted in right leads

ü Electrophysiology of Left bundle branch block

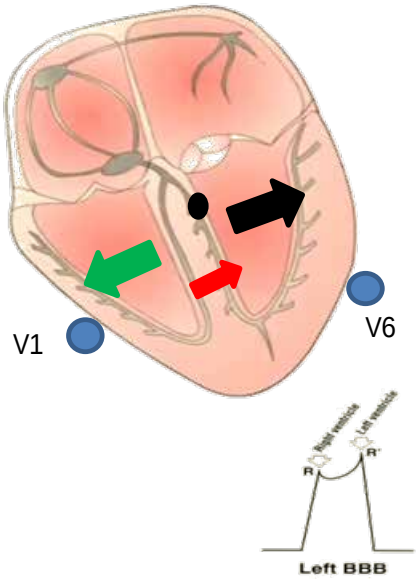
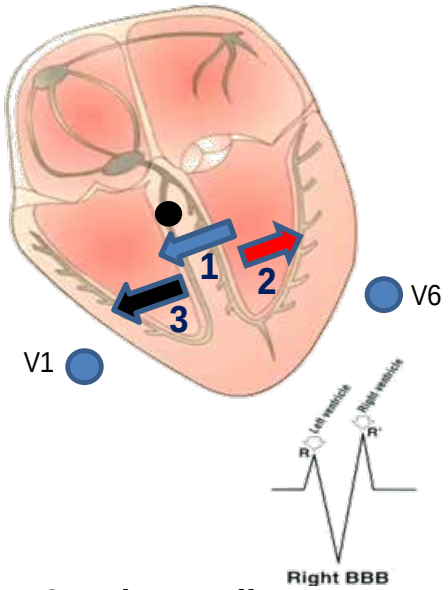
Concepts behind LBBB	illustration
Depolarization occurs on 3 phases instead of 2: - **Phase 1: -** Septal depolarization (red arrow) occurs from right to left giving: - q wave in V1-2 r wave in V5-6 **Phase 2: -** Right ventricular depolarization (green arrow is moving to right side) giving: +ve small deflection in V1 & V2 -ve small deflection in V5 & V6 **Phase 3: -** Left ventricular depolarization (black arrow is moving to left side): -ve wave in V1 and V2 +ve wave in V5 and V6	

Figure 4-8: Left BBB illustration
Quoted from *ECGteacher.com*

Concepts behind RBBB	illustration
Depolarization occurs on 3 phases instead of 2: - **Phase 1: -** Septal depolarization occurs from left to right giving: - r wave in V1-2 q wave in V5-6 **Phase 2: -** Left ventricular depolarization: - S wave in V1 & V2 R wave in V5 & V6 **Phase 3**: - right ventricular depolarization giving: - Slurred R in V1 and V2 Slurred S in V5 and V6	 **Figure 4-9:** Right BBB illustration

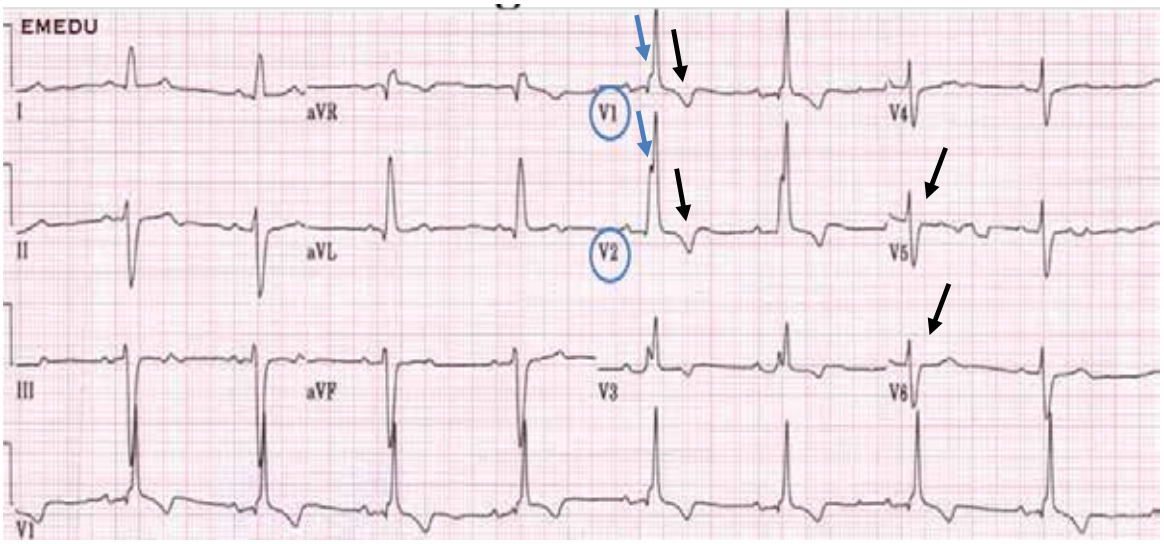


Figure 4-10: Right Bundle branch block
Notched R wave in V1 and V2 with inverted T wave, also rS pattern in V5 and V6 confirms diagnosis
Quoted from *emedu.org*

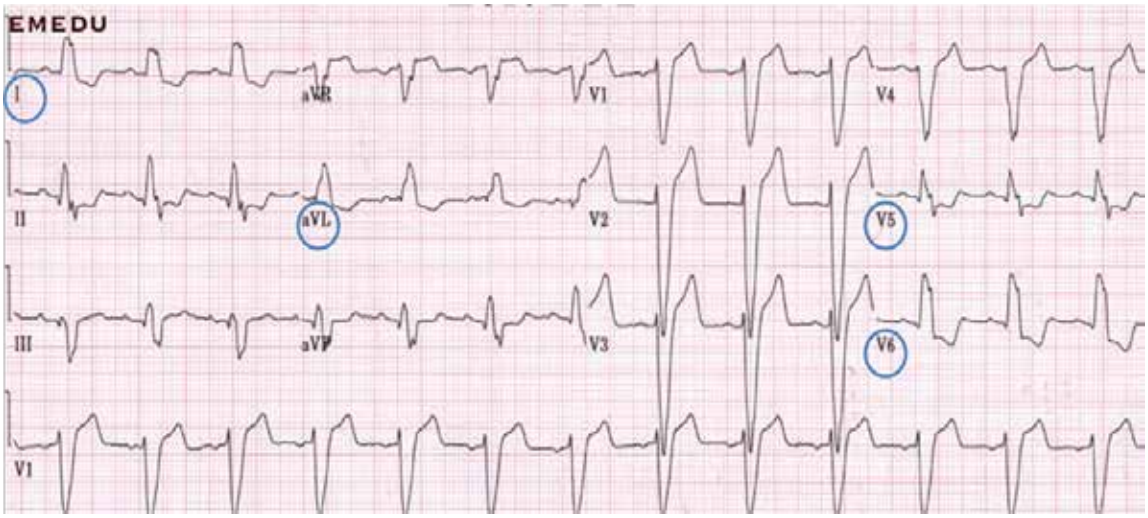


Figure 4-11: Left Bundle branch block
Notched R waves in L1, aVL, V5 and V6 + inverted T wave. rS pattern in V1 and V2 as well.

5) Ventricular hypertrophy

ü **Left ventricular hypertrophy** **Any** of the following criteria regarding QRS complex amplitude: -

1- R in V5 or V6 > 5 BS (big squares)
2- R + S in any V lead > 9 BS
3- S of V1 or V2 + R of V5 or V6 > 7 BS
4- aVF = 4 BS
5- aVL > 11 SS (small squares)

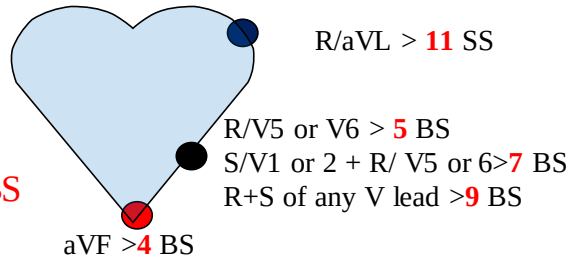


Figure 4-12: Memorize LVH

ü **LV Strain pattern: -** T wave inversion and ST segment depression in left lateral leads

for easy memory **(above figure 4-12)** **(4 5 - 7 9) large squares** (11) small squares.
The near the lead to ventricle, the higher amplitude is needed for diagnosis & vice versa.
In order from near to far: V5- V6 **(5 BS)** - aVF **(4 BS)** – aVL **(11 ss).**

ü Right Ventricular hypertrophy

V1

1- R/S ratio >1 (R larger than S) **(more sensitive)**
2- R alone 7 SS (small square).
4- aVR > 5 SS.
5-

ü **RV Strain pattern**: ST depression & / or T wave inversion in V1 & V2.

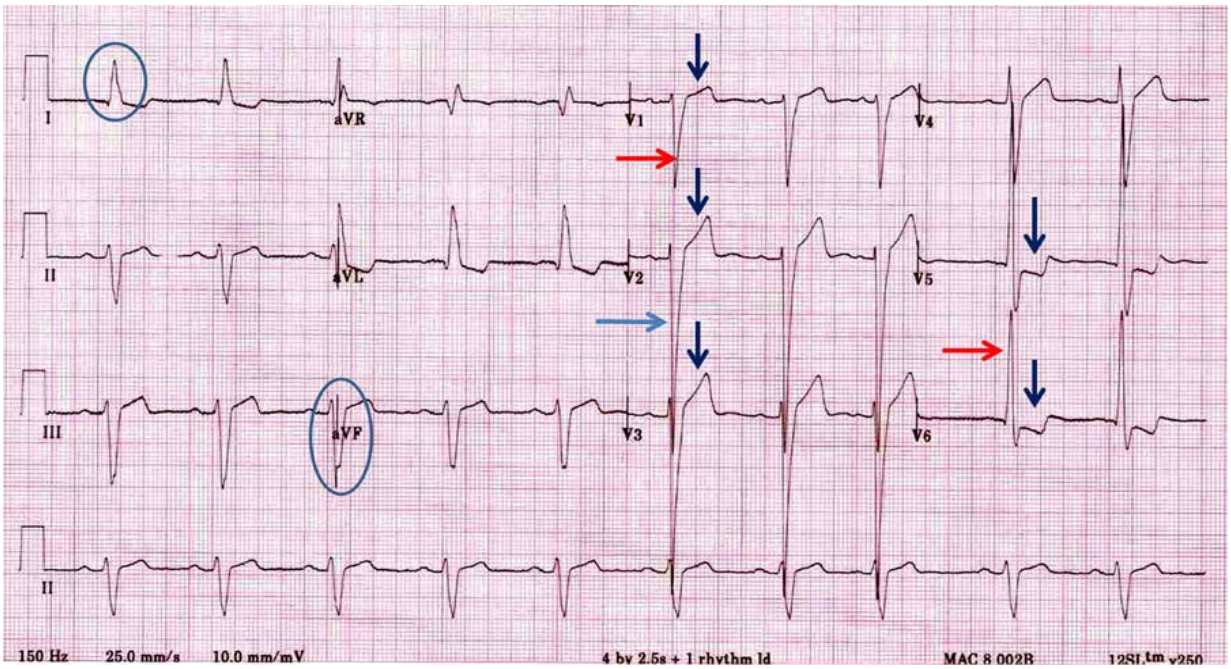


Figure 4-13: Left ventricular hypertrophy with strain pattern
LAD. r+S wave V2 >9 LS. Also S/V1+R/V6 over 7 LS, Other criteria are included.
Quoted from *emedu.org*

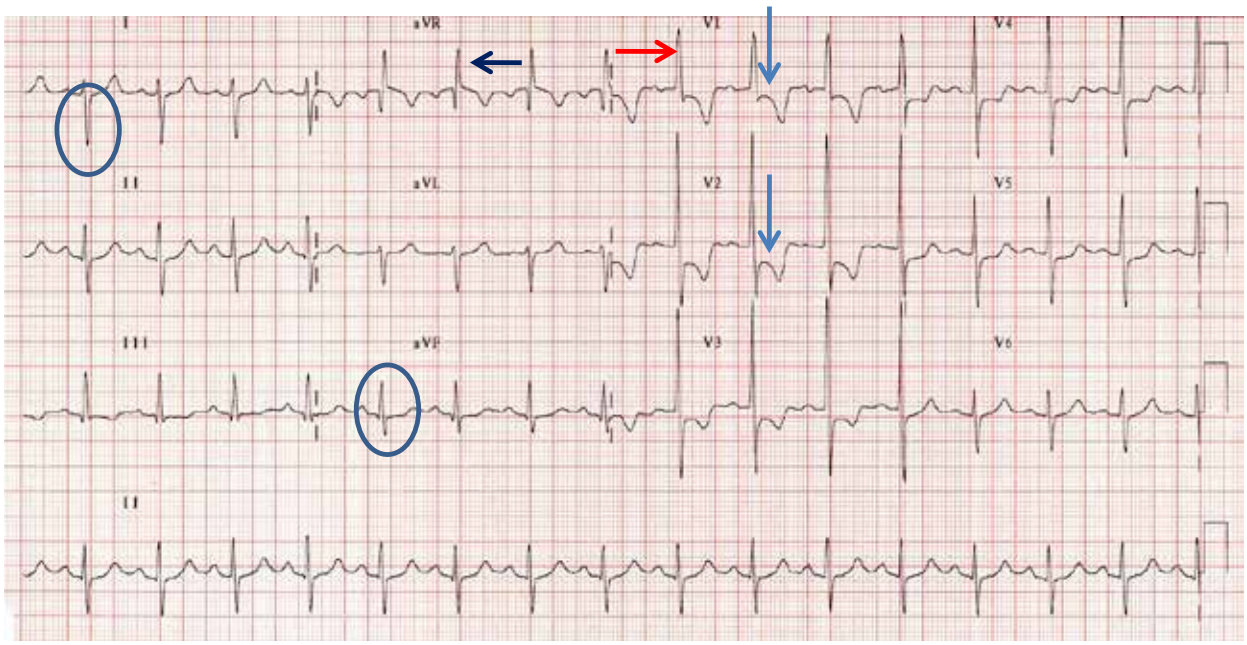


Figure 4-14: Right ventricular hypertrophy with strain pattern
RAD + R / S ratio in V1 (red arrow) >1. other criteria also included
Quoted from *lifeinthefastlane.com/*

Electrophysiology

1- An enlarged ventricle needs higher electrical impulses sufficient to be stimulated.
2- As left ventricle normally has larger muscle mass so LVH represents exaggeration of the normal state so normal R waves will be taller in left sided leads (V5, V6, aVF&aVL) while normal S waves will be deeper in right sided (V1 and V2).
3- RVH represents the reversal of normal state (S wave instead of R in left sided leads V5&V6) while R wave instead of S in V1&V2)
4- R wave amplitude in LVH will be the tallest in leads so close to LV (V5&V6). This amplitude will be lower in leads far from LV (explained before).

6) Hemiblock (Fascicular block)

Anatomy: AV bundle divides into right and left branches. The left bundle further divided into 3 fascicles: septal, anterior and posterior fascicle (Figure4-7, P31).

Table 6: Anterior and posterior hemiblock characters

Left anterior hemiblock	Left posterior hemiblock
Deep S wave in inferior leads	Deep S wave in lateral leads
Left axis deviation	Right axis deviation

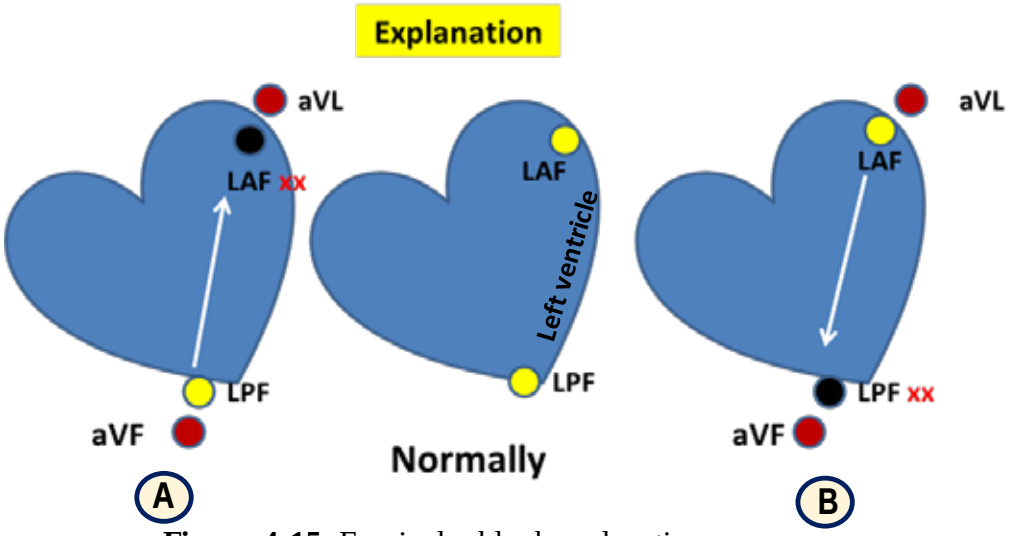


Figure 4-15: Fascicular block explanation
A: left anterior fascicular block **B:** left posterior fascicular block

Electrophysiology

Normally: - Left anterior fascicle depolarizes anterior wall in a downward & right direction while posterior fascicle depolarizes posterior wall in an upward & left direction

Left anterior hemiblock: the posterior fascicle will depolarize anterior wall in upward (away from inferior leads so negative S wave) and left direction (so left axis deviation)

Left posterior hemiblock: the anterior fascicle depolarizes posterior wall in downward (away from lateral leads so S wave) and right (so right axis deviation)

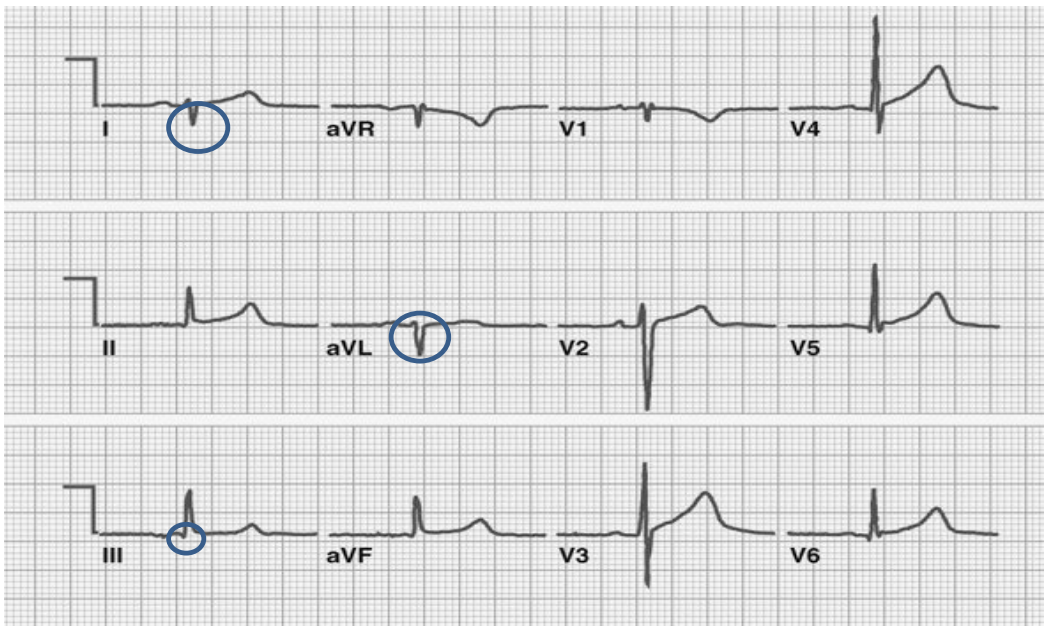


Figure 4-16: Left Posterior hemiblock
S wave in L1 and aVL + r waves in L3 + right axis deviation (RAD)
Quoted from *lifeinthefastlane.com/*

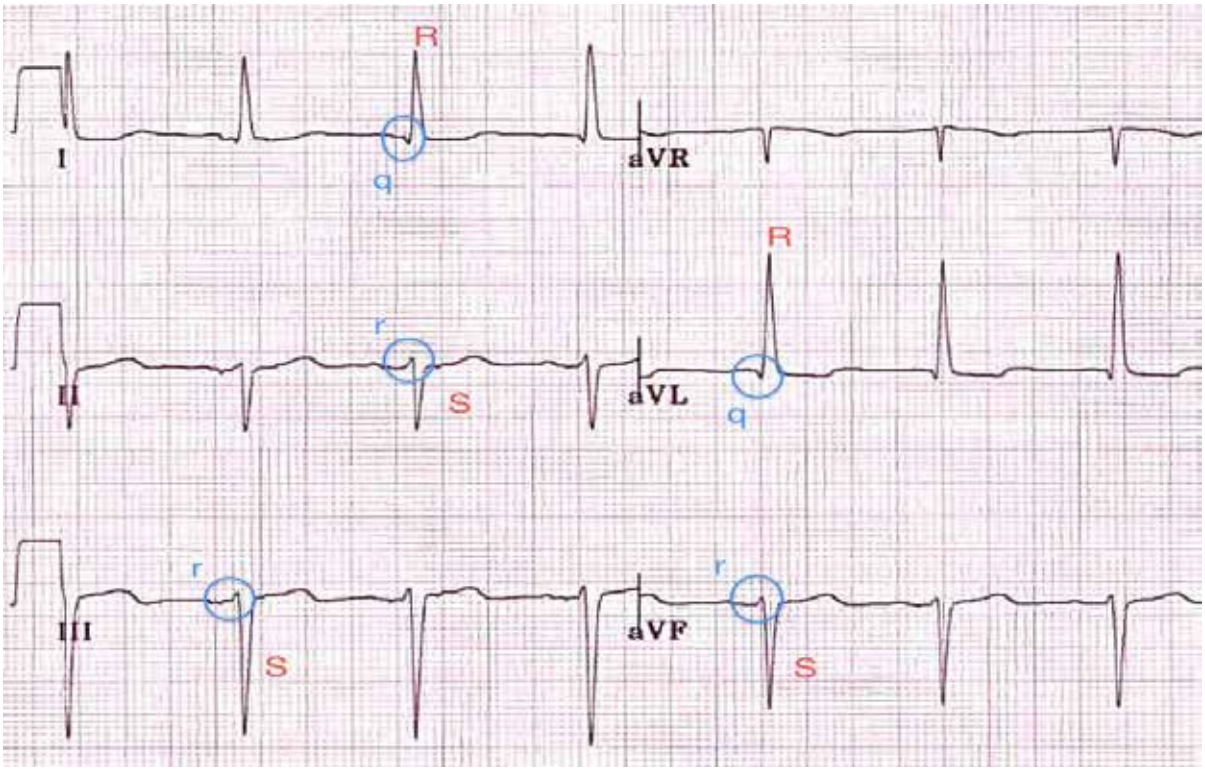


Figure 4-17: Left Anterior hemiblock
S wave in inferior leads + R wave in L1 & aVL + left axis deviation (LAD).
Quoted from *lifeinthefastlane.com*/

Clinical pearls

1- Exclude inferior wall MI in any 40 years or older patient presented with epigastric pain.

2- Right ventricular infarction is a preload dependent (due to low contractility of right ventricle). For this reason, preload reducing agents such as nitrates are contraindicated as this will lower cardiac output so worsens the condition.

3- Strain pattern reflects relative subendocardial ischemia or primary repolarization abnormalities and their presence carries higher risk of cardiovascular complications and mortality rate as well.

Chapter 5: Arrhythmia

Contents

1) Premature beats

- Premature beat are beats which come earlier than expected.
- As this beat comes earlier than a predicted sinus beat (which should be normally found instead of premature), it cancels that sinus beat with development of a pause.
- 3 types: atrial, junctional and ventricular premature beats

Table 7: Differences between premature beats

	APC	JPC	VPC
P wave	1-Abnormal shape (may be fused or buried in complex) 2-Axis may be inverted	1-Abnormal shape Inverted axis 2-Short PR interval	Absent
Pause	Non compensatory pause (RR interval around APC is not half the normal)	Non compensatory pause	Full compensatory pause (RR around it is double the normal)
QRS complex	Normal	Normal	Wide dysmorphic
Concordance	Normal	Normal	lost

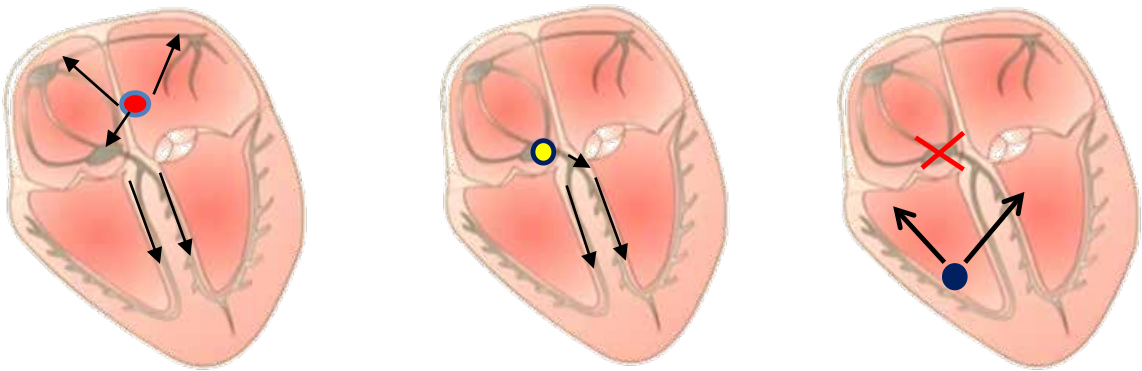


Figure 5-1: Consequences of different types of premature beats
Quoted from *ECGteacher.com*

PVCs carry risk of development of fatal polymorphic ventricular tachycardia

Electrophysiology (**Figure 5-1**):

Any wave reaches SAN would cancel a sinus beat and forms to a pause

1- APC: -

- Ø Atria are depolarized via myocardium producing abnormal shape P wave. Inverted P occurs if the focus is down in location in the atrium (low atrial focus).
- Ø This atrial wave resets SAN changing its normal rate and replaces a sinus beat. After the APC, non-compensatory pause is developed (RR interval of two sinus beats before and after APC is not double the normal RR interval between any 2 successive sinus beats).
- Ø As wave proceeds via AVN, ventricular depolarization and repolarization are not affected (hence normal complex and T)

2- JPC: -

- Ø Atria are depolarized in retrograde direction via myocardium (so abnormal P wave with inverted axis), the timing may differ that atria could be depolarized before, with or after complex (so P wave may be before complex or buried inside it or after complex). Note that the focus is inside AVN so PR interval is short
- Ø This atrial wave resets SAN giving non-compensatory pause as well.
- Ø Ventricles are depolarized via normal pathway hence normal complex and T wave.

3- VPC: -

- Ø Ventricles are depolarized abnormally via myocardium giving abnormal shape and wide QRS complex.
- Ø Due to reversed ventricular activation, repolarization will be in abnormal reversed direction so inverted T wave in +ve complex and upright T wave in -ve complexes known as loss of concordance.
- Ø No retrograde atrial activation (so absent P wave) and no SAN reset (so full compensatory pause RR of sinus beats around VPC is double the normal).

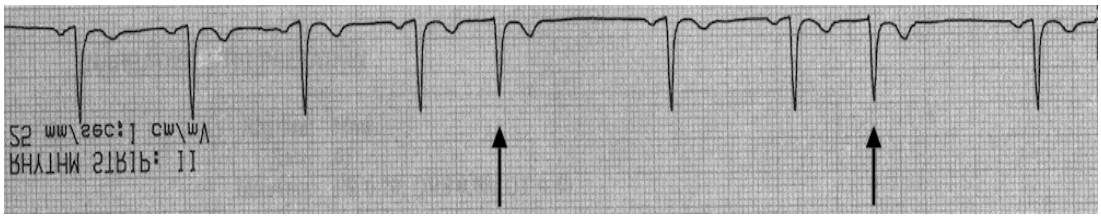


Figure 5-2: Junctional premature beat

Premature beat + narrow complex + totally absent P wave

Quoted from lifeinthefastlane.com/

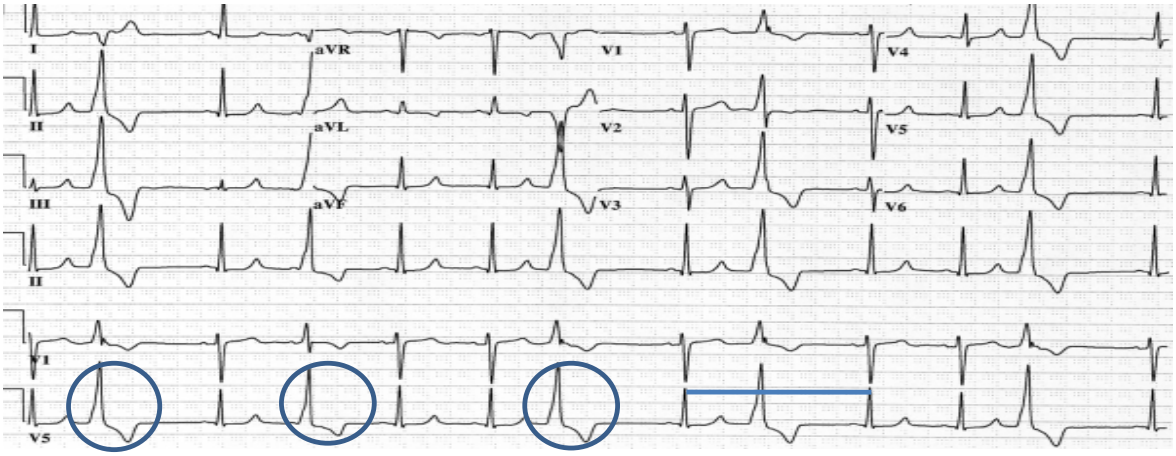


Figure 5-3: Monomorphic ventricular premature beat

It means only one ventricular ectopic beat is firing

Note the presence of full compensatory pause (blue line), wide dysmorphic complex with inverted T wave

Quoted from *www.ipej.org/*

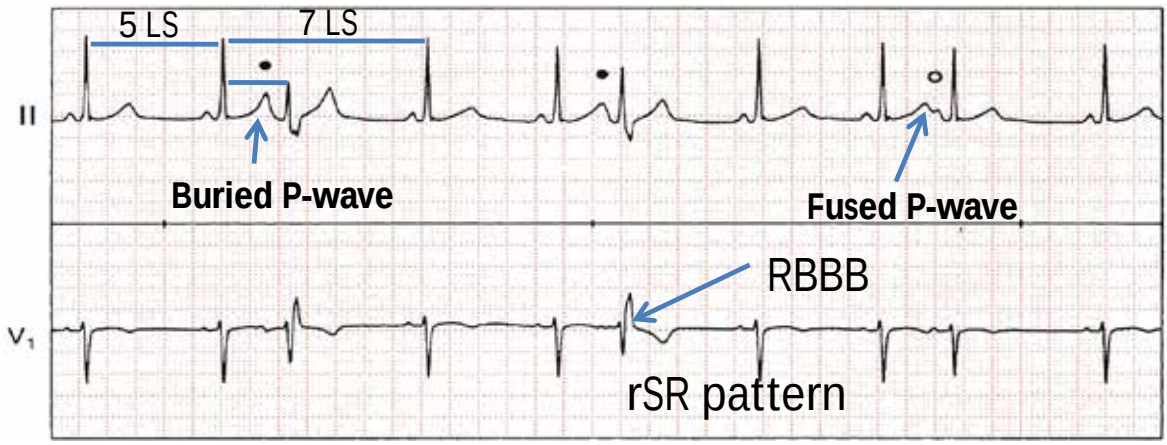


Figure 5-4: - Atrial premature beat with apparent RBBB

Notice fused P wave + non-compensatory pause

2nd beat at LII has large T wave (P wave of next beat is buried in T of previous one)

rSR pattern occasionally happened so (apparent RBBB)

Quoted from *clinicalgate.com*

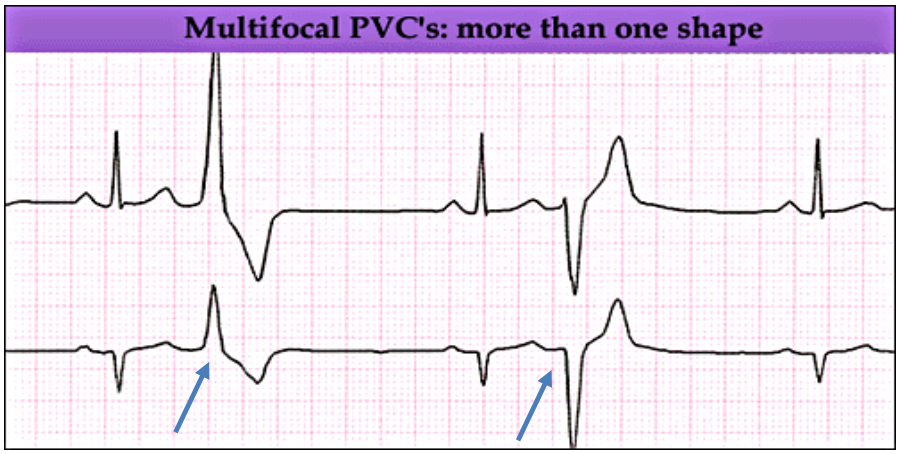


Figure 5-5: Multifocal PVCs

2nd and 4th beats came earlier than normal; both have different shapes complexes with loss of concordance

Quoted from *ward.wiki.com*

2) Supraventricular (narrow complex) tachycardia

Means tachycardia originating above the level of ventricles includes: -

Sinus tachycardia (Figure A)

1- Normal shape and axis P wave (P is inverted in aVR).
2- Normal P-R interval.
3- Narrow complex and normal T.

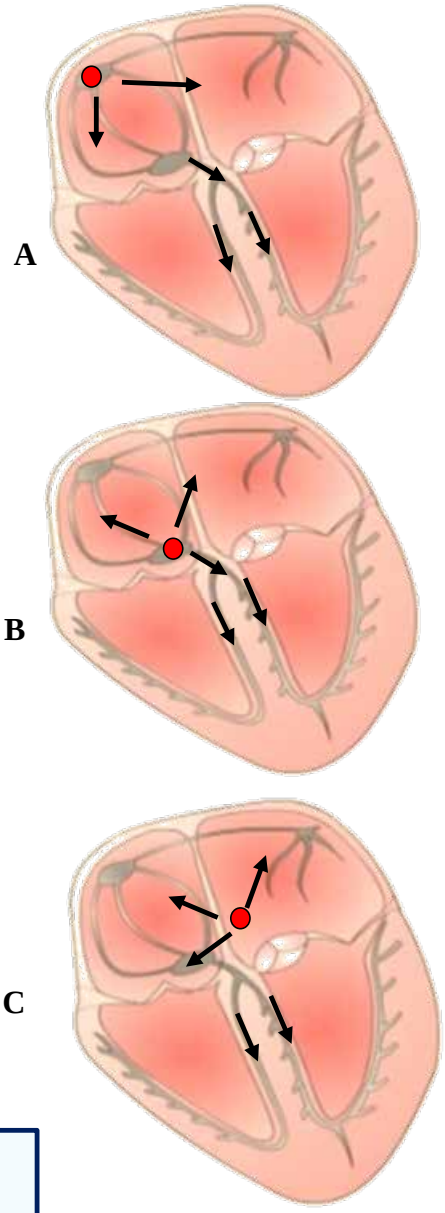
Junctional tachycardia (Figure B)

1- Abnormal shape and axis of P wave (directed superiorly) or absent.
2- Short PR interval.
3- Narrow complex and normal T.

Atrial tachycardia (Figure C)

1- Abnormal shape and variable axis of P wave (normal or inverted).
2- Normal P-R interval.
3- Narrow complex and normal T.

N.B Sometimes SVT with high rate may cause 2ry T wave and ST segment changes owing ionic disturbances resulted from tachycardia.



Figures 5-6: SVT origin and directions
A: Sinus, B: Junctional C: Atrial
Quoted from *ECGteacher.com*

Explanation of these changes are the same way as premature beats, just follow activation directions illustrated in figures 6-6 starting from red circles.

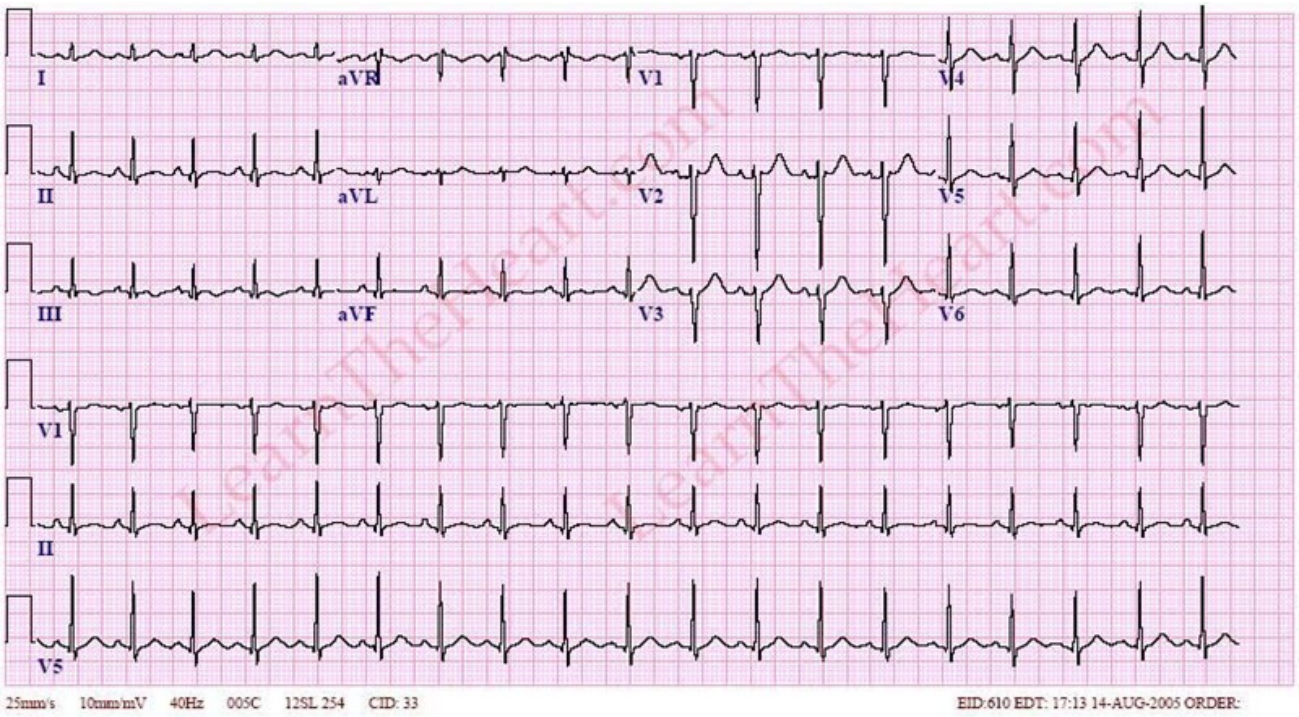


Figure 5-7: Sinus tachycardia
Normal P wave + normal PR + narrow complex + heart rate 120
Quoted from *www.healio.com/cardiology*

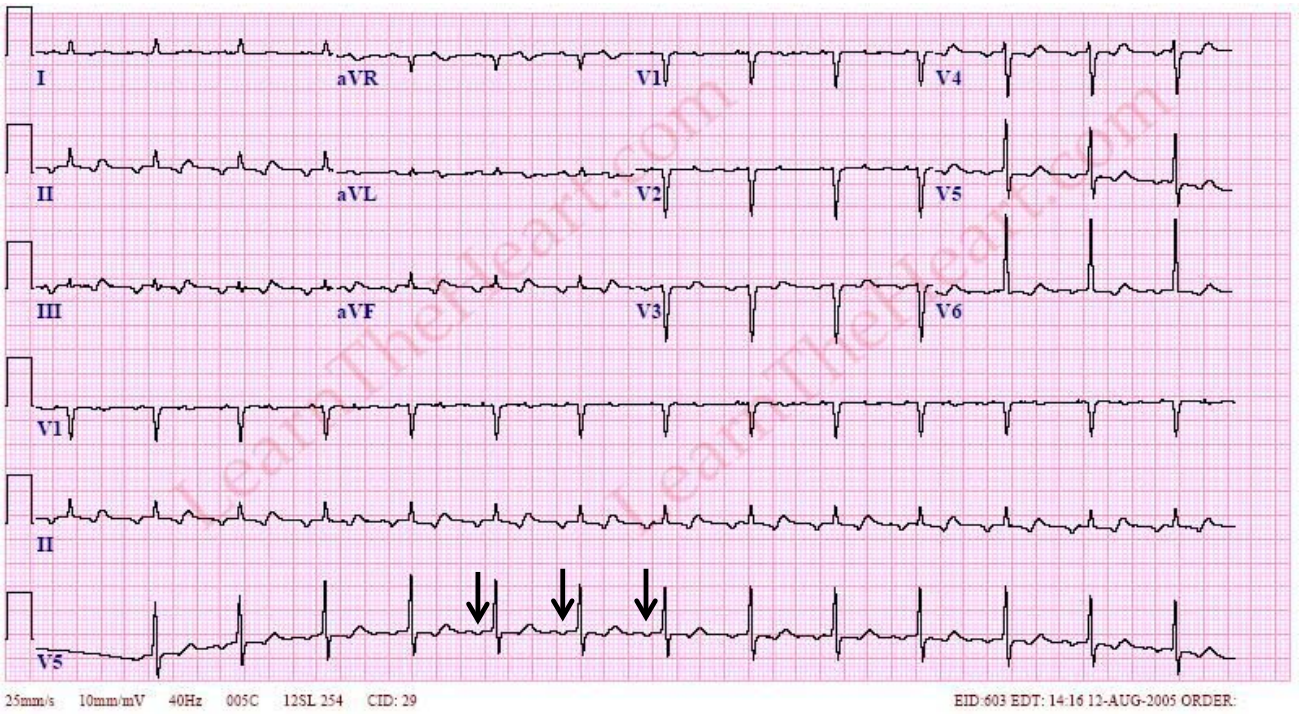


Figure 5-8: Atrial tachycardia
Inverted axis P (black arrow) + narrow complex + normal PR interval
Quoted from *www.healio.com/cardiology*

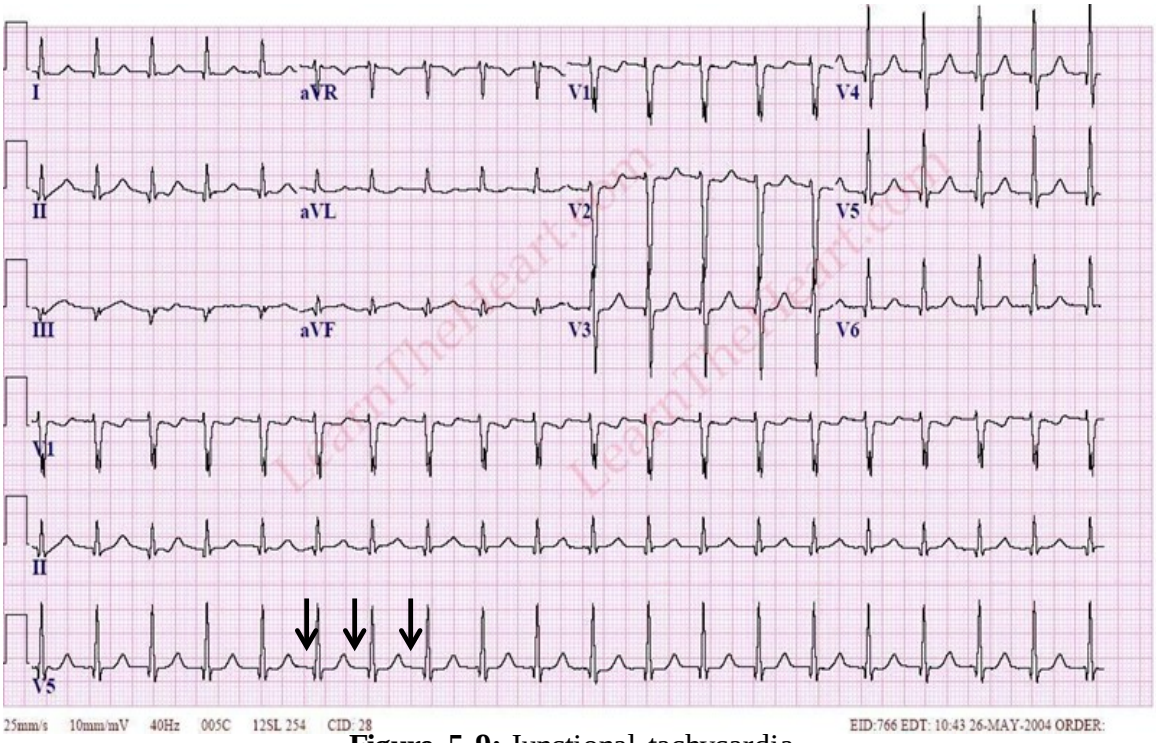


Figure 5-9: Junctional tachycardia
Absent P (black arrow) + narrow complex
Quoted from *www.healio.com/cardiology*

N.B: If tachycardia of junctional type > 140 BPM it is called AVN reentry tachycardia.

3) Atrial flutter

1- Atrial rate is usually double the ventricular rate (250-350) with regular rhythm
2- P wave: uniform shape, may take saw-tooth with inverted axis at inferior leads (flutter waves)
3- PR interval: constant
4- It is usually caused by single re-entrant in counter clockwise direction

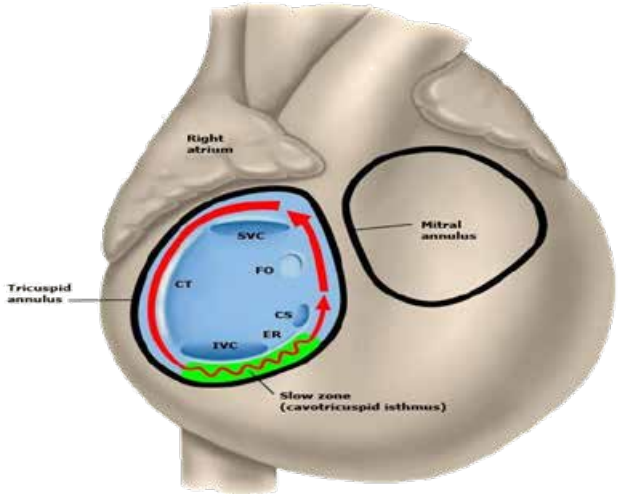


Figure 5-10: Atrial flutter typical circuit
It involves tricuspid annulus (red arrow) and cavotricuspid isthmus (green zone)
The direction is upwards (opposite to inferior leads) so flutter waves have typically inverted axis.
Quoted from *www.uptodate.com/contents/overview-of-atrial-flutter*

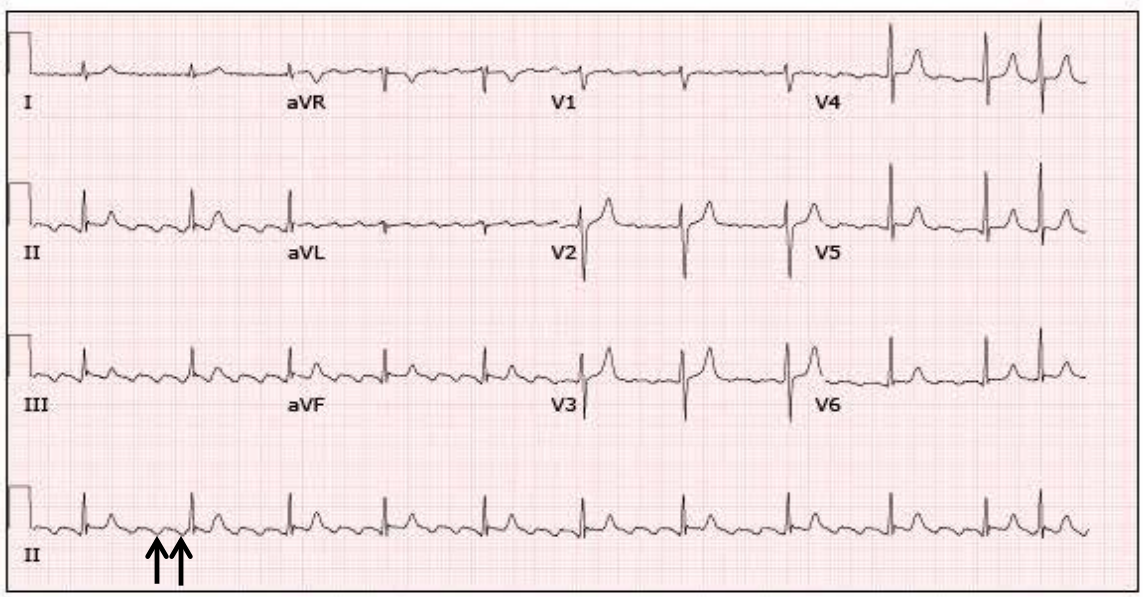


Figure 5-11: Atrial flutter with 2:1 conduction

Note inverted flutter waves (black arrows). One beat is propagated from every 2 flutter beats

Quoted from *Dr.Prithvi Puwar www.slideshare.net/DrPrith/atrial-flutter-afl-management-principals*

4) Atrial fibrillation

1- Instead of P waves, there are fibrillatory waves, no recognized repetitive pattern
2- Atrial rate exceeds 350 BPM with irregular rhythm
3- Ventricular rate usually 90-170 BPM in absence of known organic AVN disease
4- Narrow QRS complex unless abnormal conduction exists

It is usually caused by multiple micro re-entrant circuits.

NB: Atrial flutter tends to be have regular rhythm (due to single origin) unlike AF which has irregular rhythm (due to multifocal origin)

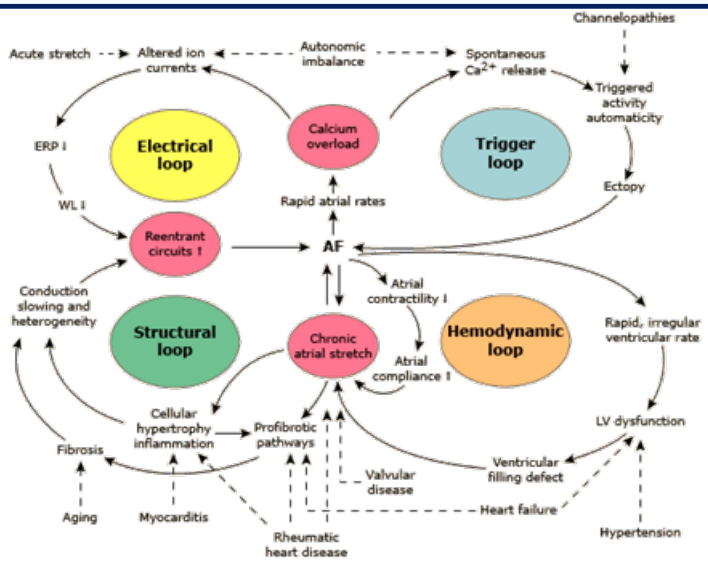


Figure 5-12: Atrial fibrillation micro-circuits

It involves several electrical, structural, trigger and hemodynamic loops

Quoted from *Ulrich et al., Pathophysiological Mechanisms of Atrial Fibrillation: A Translational Appraisal. Physiol Rev January 2011*

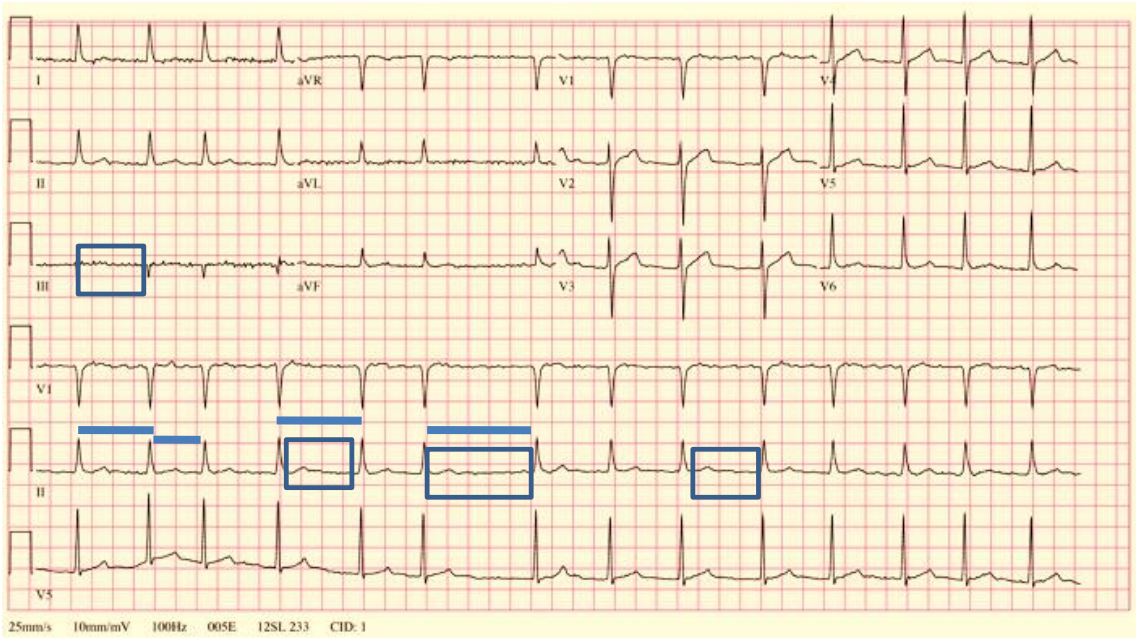


Figure 5-13: Fine atrial fibrillation

P waves are replaced by fibrillatory waves

The more the number of circuits discharging the less will be the fibrillatory waves amplitude

Quoted from *dxline.info/diseases/atrial-fibrillation*

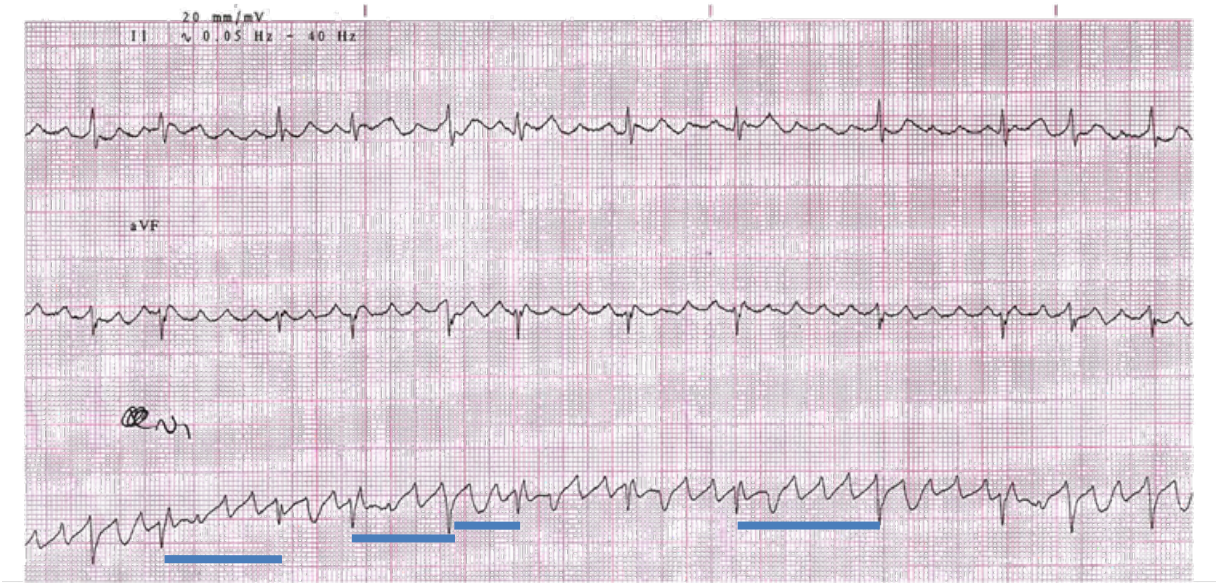


Figure 5-14: Coarse atrial fibrillation

You will be tricked by the shape of waves thinking they are flutter waves.

Extremely irregular rhythm and baseline twisting favors fibrillation over flutter

Quoted from *Dr-Alberto Giniger, ECGpedia.org*

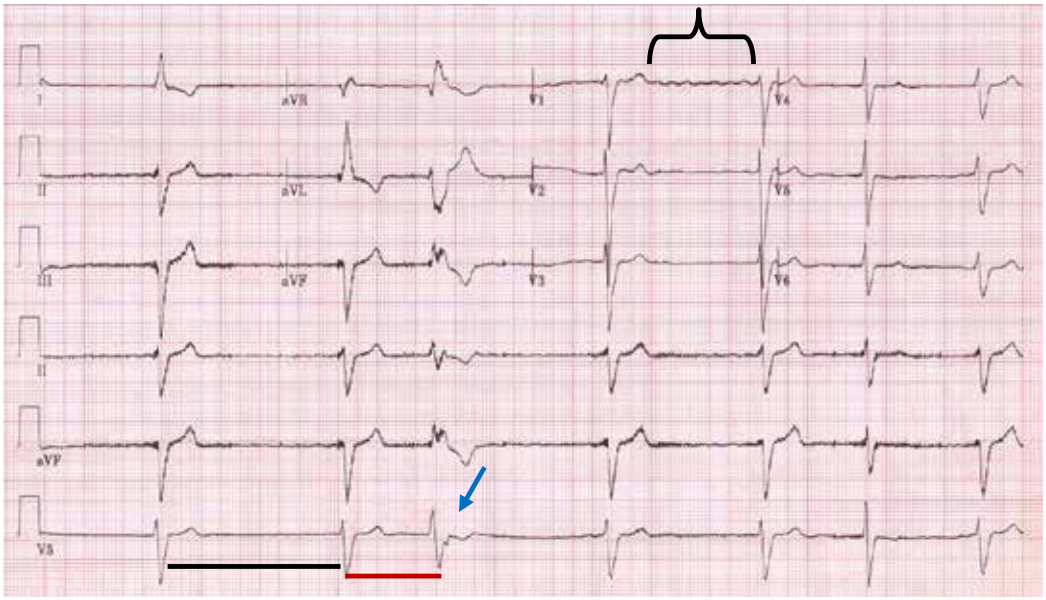


Figure 5-15: Atrial fibrillation with Ashman's phenomenon
Fibrillation waves are evident in V1 lead (marked above)
The beat with abnormal complex (blue arrow) came after long beat (black line) then short beat (red line); Ashman's phenomenon
Quoted from *lifeinthefastlane.com*

Ashman's phenomenon: An aberrant ventricular conduction due to change in QRS cycle length (aberrancy means deviated from normal pathway).

- It is most commonly found in **atrial fibrillation**.
- **Electrophysiology:** Changes in heart rate are associated with concomitant changes in refractory period of conducting system.

  When a cycle with slow rate (**black line** in figure 5-15) changes into a cycle with rapid rate (**red line** in same figure), the beat of the short cycle (**blue arrow**) founds bundle branches in the absolute refractory period so this impulse will be blocked at the level of bundle branches giving BBB morphology.

5) Ventricular tachycardia

1-Wide dysmorphic complexes
2-T wave opposite to QRS complex vector
3-Evidence of AV dissociation (**may** be found so supports diagnosis)

- Capture beat (normal sinus wave occasionally exist).
- Fusion complex (complex formed partially by sinus beat and by ventricular origin).
- Dissociate P wave.

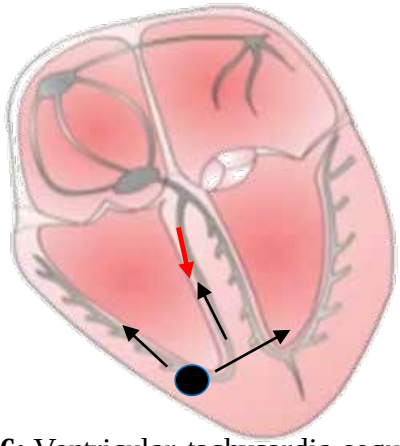


Figure 5-16: Ventricular tachycardia sequences of activation
Red line with black one at the level of bundle branch gives fusion complex
Quoted from *ECGteacher.com*

Electrophysiology

1- The concept of wide dysmorphic complex is based on abnormal origin and pathway for ventricular depolarization. As a result, repolarization direction is also altered making T wave opposite QRS complex vector.

2- Sometimes a sinus beat succeeds to be conducted via AVN to ventricles before the beat of ventricular focus and excites them. This beat is called **capture beat**.

3- Sometimes, a sinus beat reaches bundle branches and depolarizes them proximally. At the same time distal parts of these bundles are depolarized from ventricular focus, hence the produced complex will carry some features of normal sinus and abnormal ventricular beat (so called **fusion complex**).

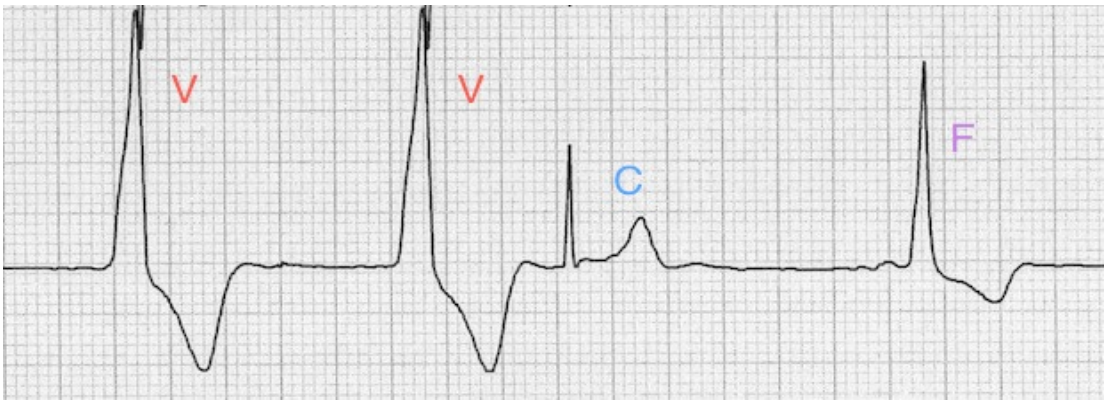


Figure 5-17: Evidence of AV dissociation
V: complex of ventricular tachycardia, **C:** Capture beat (normal sinus beat), **F:** Fusion complex (note its amplitude is midway between sinus and ventricular beat. There is T wave inversion as well)
Quoted from *blog.naver.com*

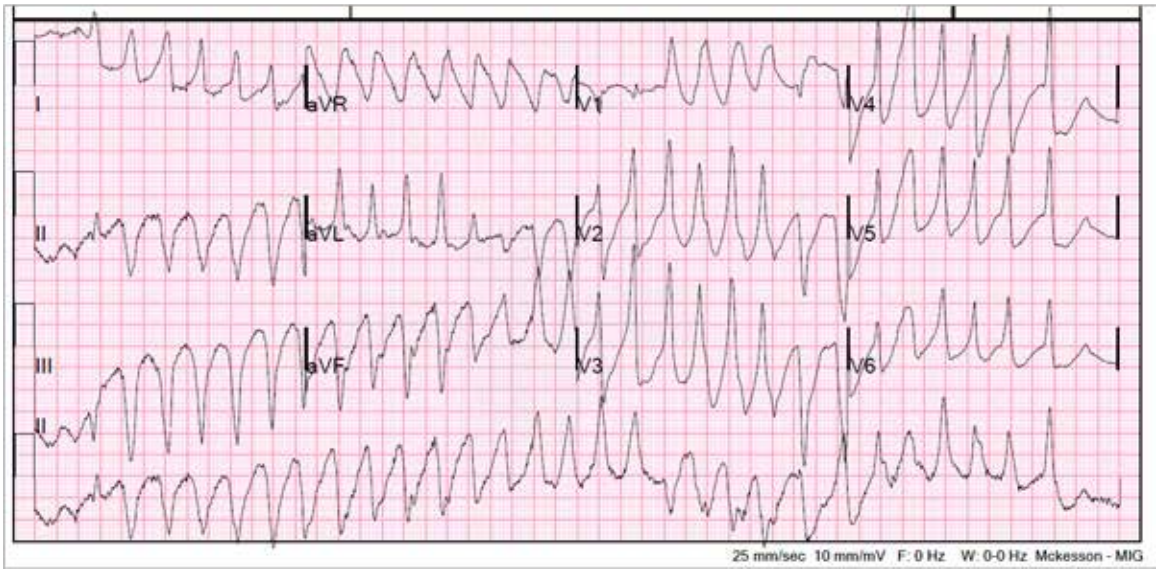


Figure 5-18: Polymorphic ventricular tachycardia
Wide complexes with absence of P wave. There are small and large complexes which mean multiple foci discharging at the same time (arrows) thus polymorphic.
Quoted from *Dr Smith's ECG blog hqmeded-ecg.blogspot.com*

6) Torsade de pointes (twisting of spikes)

- Form of polymorphic ventricular tachycardia (VT) characterized by a gradual change in the amplitude and twisting of the QRS complexes around the isoelectric line
- It is usually initiated by a premature beat with a phenomenon called R on T phenomenon (this phenomenon means PVC occurs during the preceding T wave) (**Figures 5-19, 5-20**).

7) Ventricular Fibrillation

- Extremely chaotic rhythm with very high ventricular rate which could lead eventually to cardiac arrest and death. The definitive intervention is defibrillator.
- This results from multiple reentrant circuits within ventricle.

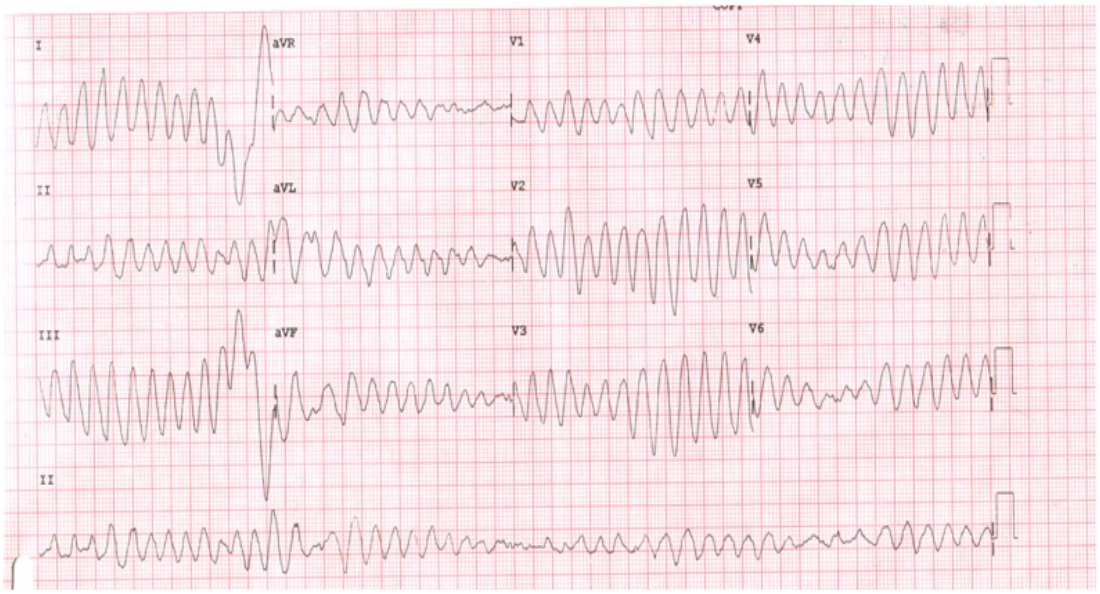


Figure 5-19: - Torsade de pointes (twisting of spikes)
Quoted from *studymedicalphotos.blogspot.com*

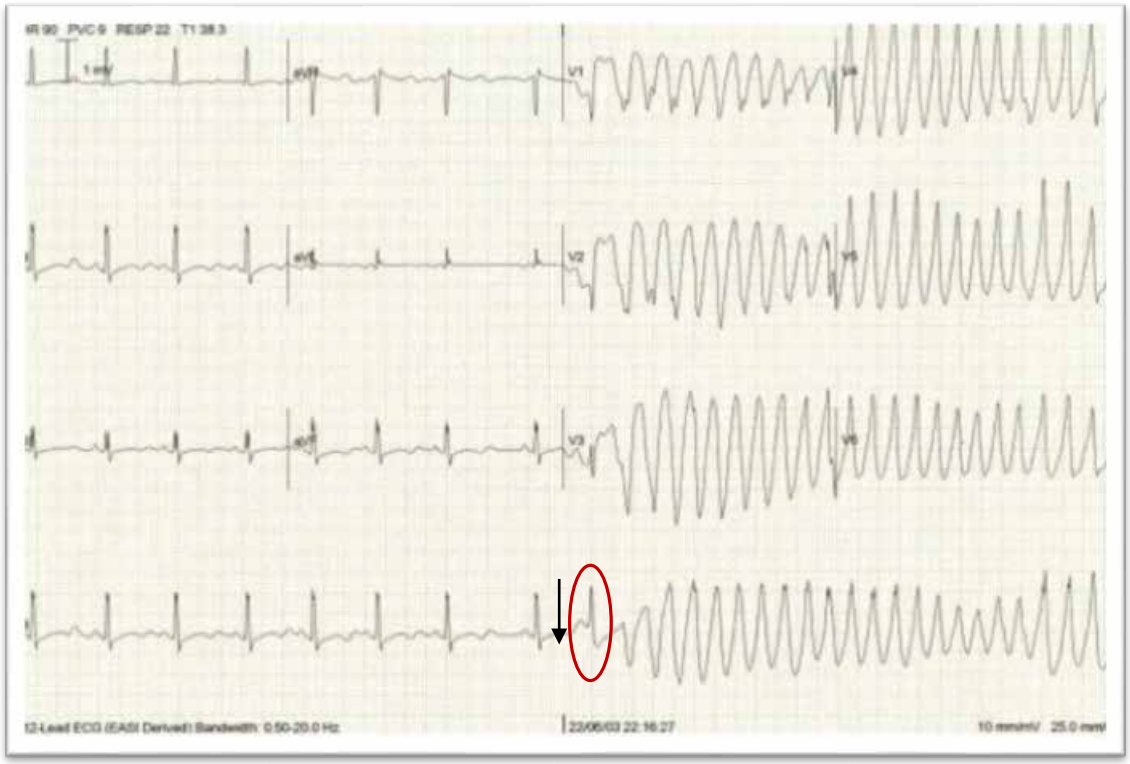


Figure 5-20: R on T phenomenon preceding Torsade
Arrow points to T wave, note that premature beat (red circle) has P wave which came directly at the end of T wave so called R on T phenomenon. This premature beat precipitates torsade de pointes
Quoted from *lifeinthefastlane.com/*

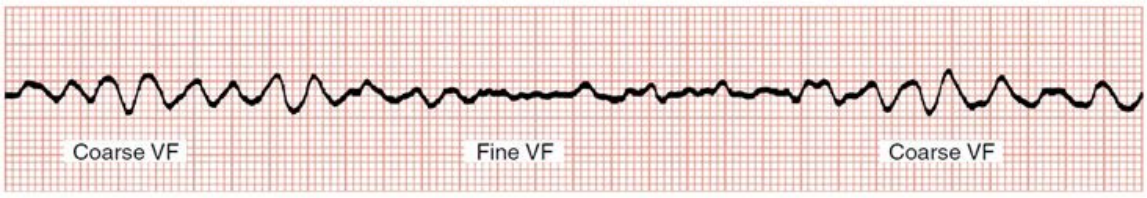


Figure 5-21: - Ventricular fibrillation
Quoted from *Goldberger's clinical electrocardiography, 8th ed*

8) Pulseless electrical activity (electromechanical dissociation)

Features: **Unresponsive** patient with no palpable pulse but still some organized activity on ECG.

Pathophysiology: - Conditions lead to failure of contraction precipitates it. These conditions are either **reduced preload** (no sufficient volume for stretching to achieve contraction), **impaired contractility** or rarely **very high afterload** against left ventricular muscle.

9) Asystole

A condition in which patient is not responsive, has no pulse and flat line on ECG. It carries worst prognosis.

Pathogenesis of arrhythmia

- ü Cardiac cycle begins with an electrical activity which is converted into mechanical one.
- ü During mechanical action, the net current is zero. This is achieved via well balanced movements of ions through the membranes.
- ü Excitation contraction interaction could be summarized as follow **(Table 8).**

Table 8: Summary of excitation contraction interaction

Action	Excitability phase	Responsible ion
Depolarization	**Rapid depolarization**	**Na influx**
Contraction	**Plateau phase (corresponds ST segment)**	**Ca influx for contractility K efflux for membrane stabilization**
Repolarization	**Rapid repolarization (corresponds T wave)**	**K efflux**
Relaxation	**Resting phase (diastolic depolarization)**	**Na-K pump, Na-Ca exchanger and Ikr channels**

** Remember phases of myocardial action potential*

Phase 0: Rapid depolarization (rapid Na influx)
Phase 1: Early partial repolarization
(K efflux vs. inactivation of Na channels)
Phase 2: Plateau phase
(K efflux vs. Ca & delayed Na influx)
Phase 3: Rapid repolarization (rapid K efflux)
Phase 4: Resting phase (Na-K pump mainly)

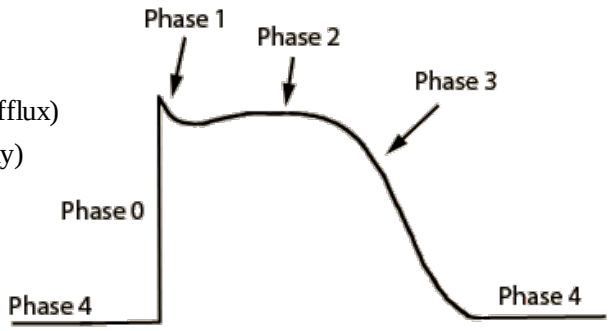


Figure 5-22: Myocardial action potential
Quoted from*theeplab.com/B-The-Members-Center/C-Cardiac-AnatomyPhysiology/*

- During resting phase of myocytes, SAN generates impulses (automaticity). This is achieved via combined Ca & Na influx and decreased K efflux within it. Automaticity is only found in pacemaker cells & myocardial cells do not capable of automaticity **under normal conditions**.
  - ü Increased Ca influx leads to **enhanced automaticity** of SAN (e.g. sympathetic response) and **abnormal automaticity** of myocytes (Digitalis toxicity)
- During plateau phase, movement of Ca in and K out makes membrane potential stabilized. Any electrical activity is produced during this phase would produce **triggered activity**, this could be achieved via: -
  - ü **Increased Ca influx** (e.g. digitalis effect or catecholamines)
  - ü **Decreased K efflux** (e.g. class IA and III antiarrhythmic agents)
- Automaticity is responsible for many conditions such as sinus tachycardia, junctional tachycardia and premature beats.
- Triggered activity is responsible for generating premature beats which could lead to a fatal tachyarrhythmia (due to reentry formation).
- Both conditions are managed medically by drugs reducing Ca such as Calcium channel blockers and beta blockers.

Reentrant theory: -

Circus movement reentry occurs when an impulse propagates around an anatomic or functional obstacle and reexcites the site of origin.

Two factors are required for re-entry formation

1- Presence of a site with two pathways of different conduction velocity (one slow conducting zone and other fast) ⟶ delaying factor

2- Premature beat

Applied example would be on AVNRT **(Figure 5-23).**

Some people have 2 different entries for AVN: -

Fast pathway: - conducting fast and recovering slowly.

Slow pathway: - conducting slow and recovering fast.

The sequences occur as follow: -

1- Premature beat (gets earlier than normal) reaches AVN when slow pathway is available for propagation (being recovered fast) while fast pathway is still unavailable (slow recovery)

2- The beat enters slow pathway, depolarizing AVN and heading towards AV bundle. During AVN depolarization, the fast pathway becomes available and able to conduct the impulse in a retrograde fashion

3- In conclusion, the impulse is propagated forwards to AV bundle and backwards towards its origin via fast pathway. This results in self- perpetuating circuit generating impulses at very high rate and taking control over the heart.

- Atrial flutter, AVNRT and AF are usually caused by this mechanism.
- For unconscious & hemodynamically unstable patients, cardioversion is appropriate to terminate the attacks of tachycardia caused by single re-entrant mechanism.

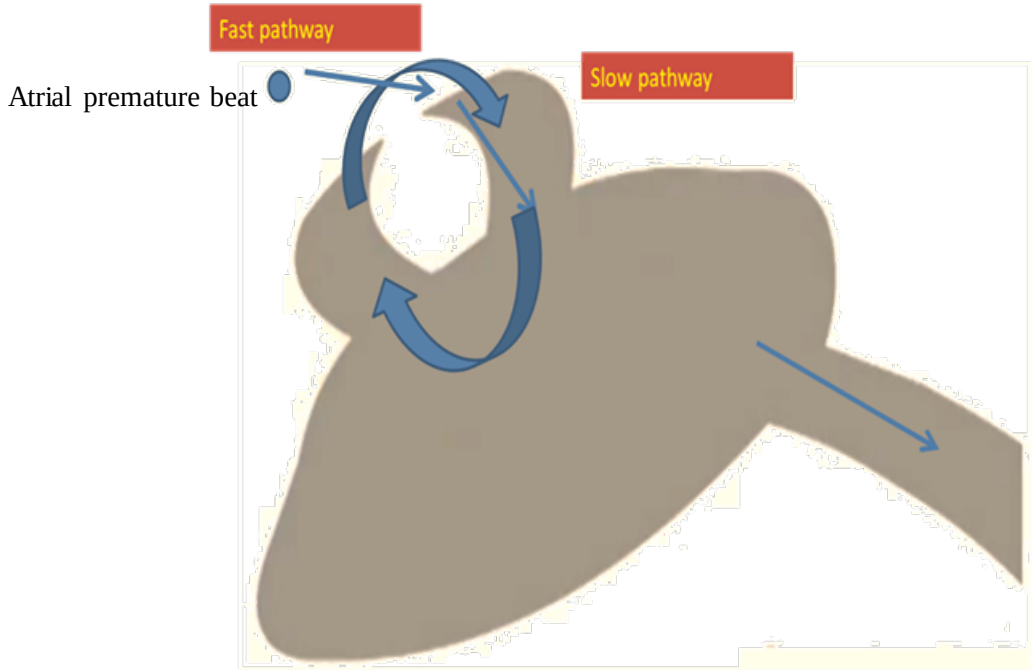


Figure 5-23: Schematic illustration of AVNRT
Quoted from *ecgteacher.com*

To summarize, 3 common identified electrophysiological mechanisms behind arrhythmia:

1- Automaticity: SAN firing increases or abnormally the myocardium of atria or ventricles begins to fire impulses as an ectopic focus. This occurs due to increased Ca as in sympathetic stimulation and digitalis toxicity.
2- Triggered activity occurs as a result of increased Ca influx or decreased K efflux during plateau phase of action potential. This phenomenon could precipitate torsade de pointes.
3- Reentry is a self- perpetuating circuit generating impulses at very high rate which leads to development of atrial and fatal ventricular tachyarrhythmia.

Clinical Pearls

1) Life threatening rhythms associated with cardiac arrest are classified into shockable and non-shockable rhythm.

- ü **Shockable rhythms** are rhythms which could be terminated by defibrillator **(which is an electrical device)** as they represent **electrical failure.**

* They include pulseless ventricular tachycardia and ventricular fibrillation.

- ü **Non shockable rhythms** are rhythms in which defibrillator cannot terminate it as they represent actually **mechanical failure** (electrical activity is found but cardiac muscle does not respond by contraction)

* These include Pulseless electrical activity (electromechanical dissociation) and Asystole. At these conditions, defibrillator has no role. Correction of precipitating factors of PEA along with using medications as epinephrine could lead to restoration of normal sinus rhythm in some cases.

2) Check for atrial flutter (AFL) for any atrial rate 200-400 BPM or ventricular rate of 150 BPM or higher.

3) AVNRT is often terminated using methods which slow conduction at AVN including vagal valsalva maneuver or adenosine (AVN blocker). Another premature beat could terminate AVNRT spontaneously.

Chapter 6: ST segment, T wave and QT interval

Contents

1) Physiological parameters

- **QT interval** Less than 0.44 sec **(**Less than 11 mm) or half of RR interval. It represents ventricular cycle (indicator for ventricular repolarization)
- **ST segment** from the end of complex (starts at J point) to beginning of T wave
- **T wave** amplitude is typically up to **5mm in limb leads** and **10 mm in chest leads**.
- **U wave** may be absent, represents papillary muscle repolarization. Appears when heart is slowing its rate, its height is **1/10** of T wave

2) Potassium disturbances

Hyperkalemia

Changes	Electrophysiology concepts
Hyperacute T wave	Increased extracellular K enhances K efflux through Ikr channels so makes repolarization amplitude high so T wave will be tall.
1-Wide then loss of P wave 2-Long PR segment 3-Wide bizarre QRS complex 4-Bradycardia 5-Sine wave appearance	Depolarization is initially increased (via membrane depolarization) then suppressed as hyperkalemia inactivates Na channel so slows conduction in atrial and ventricles.

Increased K outside the cell leads to decreased K efflux during rest through leaky K channels thus more +ve charges are trapped inside the cell elevating resting membrane potential (eg, -70 to -40) which means membrane depolarization.

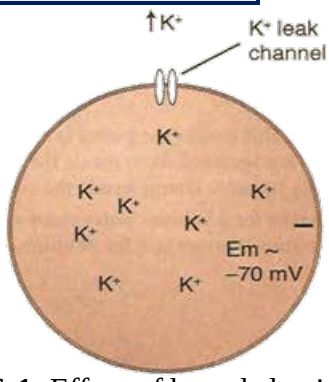


Figure 6-1: Effect of hyperkalemia on membrane potential
Quoted from Kaplan physiology book 2013

* For revision of myocardial action potential, refer to P.53
* IKr channels are K channels which are sensitive to serum K level (K efflux increases with high serum K levels and decreases with low serum K).

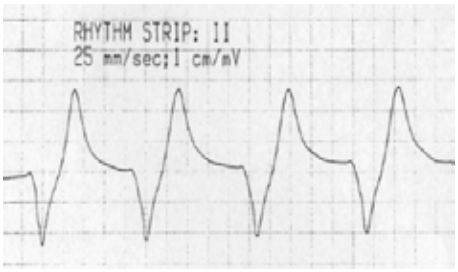


Figure 6-2: Sine wave of severe hyperkalemia
Quoted from lifeinthefastlane.com

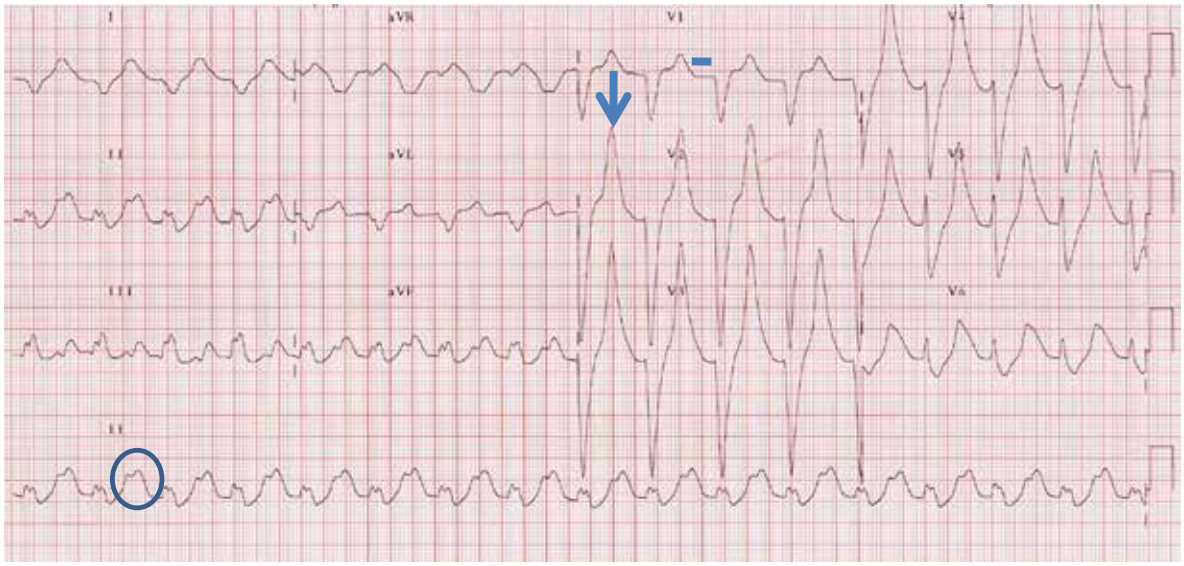


Figure 6-3: Hyperkalemia
Note peaked T wave (arrow) + bizarre QRS morphology (circle) and long PR segment (line)
Quoted from *lifeinthefastlane.com*

ü Hypokalemia

Changes	Electrophysiologic concepts
T inversion, flattening Appearance of U wave Long QU interval	Hypokalemia causes decreased K efflux via IKr channels , thus repolarization takes longer duration to be completed . This makes T wave more flat or even inverted if hypokalemia is enough to reverse the direction of repolarization. Slow rate will result in U wave appearance
Wide tall P wave , long PR interval	hypokalemia causes membrane hyperpolarization so net result is delayed depolarization.

Hypokalemia causes increased K efflux across membrane during rest , the net result is that more negative charges are trapped inside the cell , making membrane potential more negative (e.g -70 to -100) (hyperpolarization state).

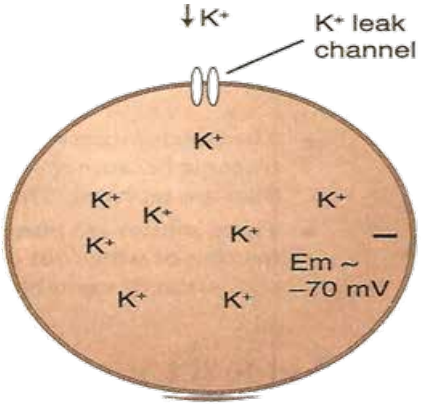


Figure 6-4: Effect of hypokalemia on membrane potential
Quoted from *Kaplan physiology book 2013*

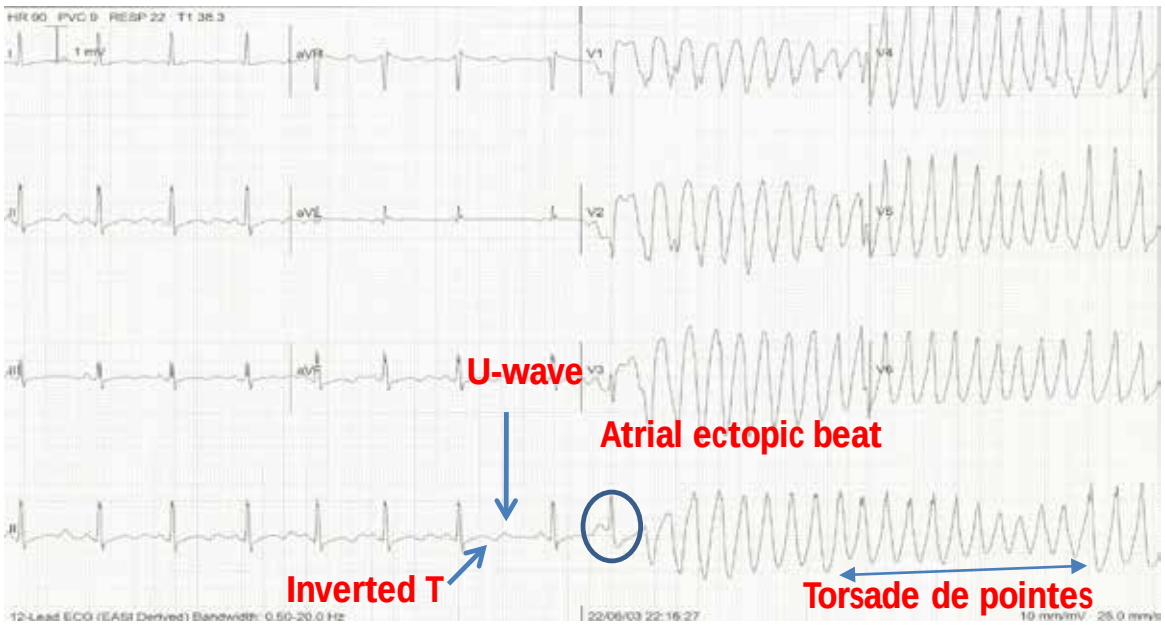


Figure 6-5: Hypokalemia
Inverted T wave + U wave. Also an atrial premature beat has initiated torsade des pointes with R on T phenomenon.
Quoted from *lifeinthefastlane.com*

3) Ca disturbances

Normal Ca level: - 9-11 meq/L

Table 9: Features of hypercalcemia and hypocalcaemia.

Hypercalcemia	Hypocalcemia
Short ST segment	Long ST segment
Short QT interval Severe cases cause Osborn wave	Long QT interval

Electrophysiology

1- Ca influx occurs during plateau phase of cardiac cycle thus any changes in Ca would only cause alterations in the duration this phase .

2- Plateau phase corresponds to ST segment on ECG . As long as there is no method to calculate ST segment duration , QT interval will be used as an indicator for lengthening or shortening of ST segment .

3- Hypocalcemia causes slow Ca influx thus prolonging plateau phase, ST segment and subsequently QT interval (Hypocalcemia causes Long QT interval).

4- Hypercalcemia causes fast Ca influx thus shortens plateau phase , ST segment and subsequently QT interval (Hypercalcemia causes short QT interval).

* Long QT means longer duration of repolarization . This leads to development of premature beats (via triggered activity) which could initate a reentrant mechanism between cardiac layers causing torsade de pointes .

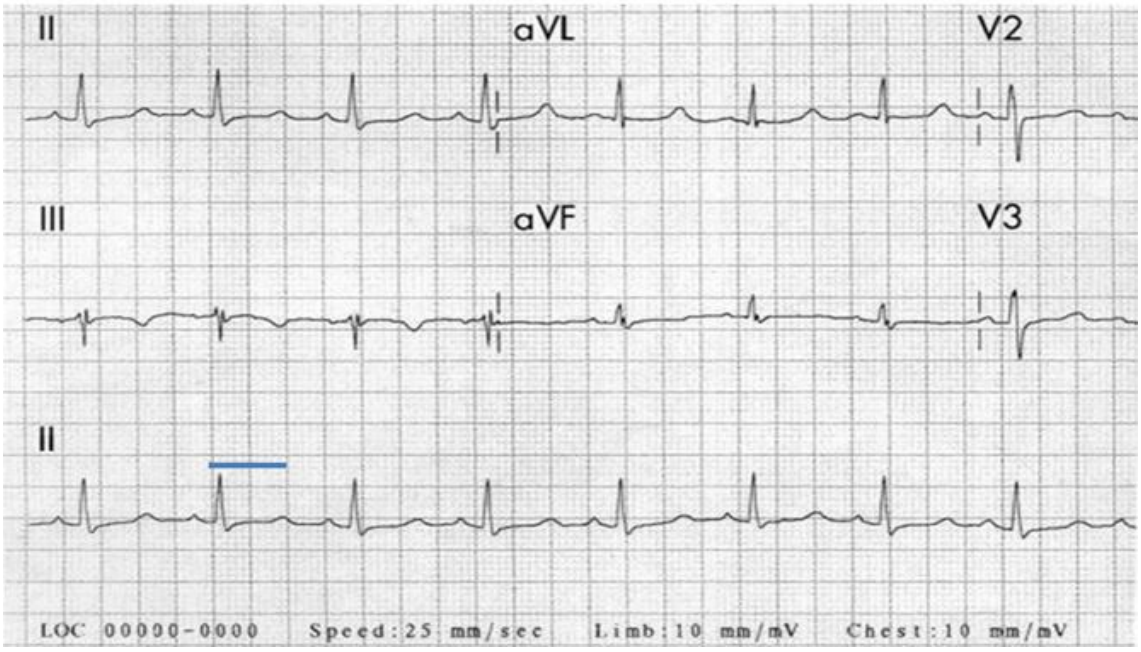


Figure 6-6: Hypocalcemia
Corrected QT interval = 0.59. The patient is known to have Digorge syndrome
Quoted from Kar et al. (2005)

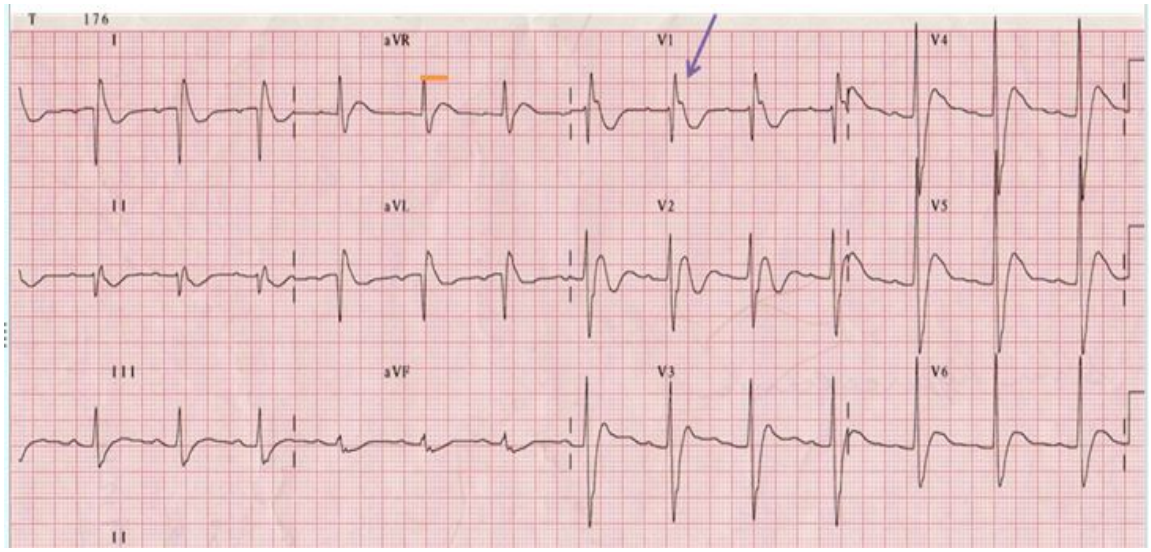


Figure 6-7: Hypercalcemia
QT interval is very short + Osborn wave (blue arrow)
This patient has parathyroid adenoma; shortly he suffered VF after this ECG
Quoted from *Dr James Hayes, FACEM*

4) Brugada syndrome

ü Definition

- Brugada syndrome is a disorder characterized by sudden death associated with one of several ECG patterns characterized by incomplete right bundle-branch block and ST-segment elevations in the anterior precordial leads
- It is a common cause of sudden death during rest or sleeping

ü Patterns

Pseudo right BBB combined with persistent ST elevation in V1 and V2 are the striking features in patients with Brugada syndrome.

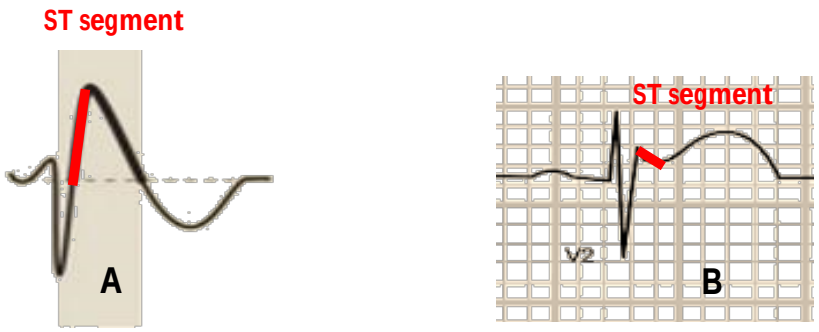
Diagnosis is only needed in one right sided precordial lead

Ø Type 1 (coved pattern)

ST segment is elevated, then descends with upward convexity into inverted T wave

Ø Type 2 (saddle back)

ST segment is elevated then descends to baseline and rises again to an upright or biphasic T wave or even inverted.



Figures 6-8: Morphology of brugada patterns
A: coved pattern **B**: saddle back
Quoted from nature.com/nrcardio/journal/vaop/ncurrent/fig_tab/nrcardio.2016.143_ft.html

Table 10: Patterns of ST abnormalities in V1 to V3 in Brugada syndrome

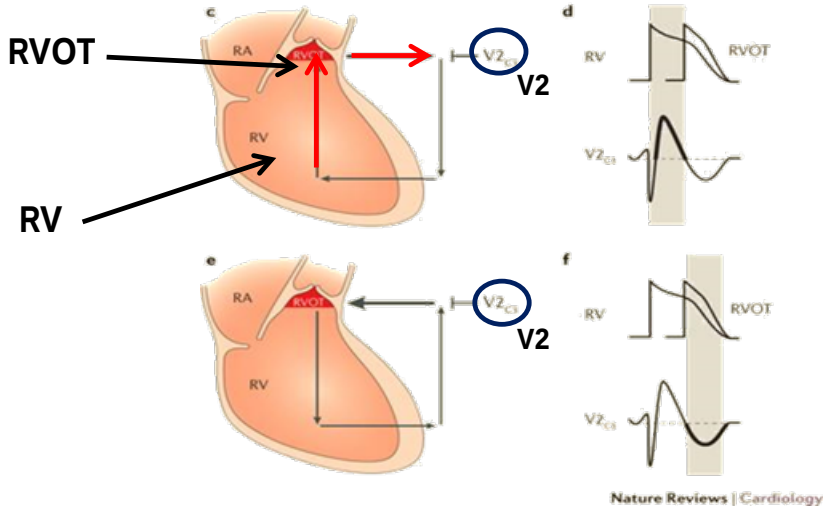
ECG changes	Type 1	Type 2
J wave	≥2mm	≥ 2mm
T wave	inverted	upright
ST-T wave	Coved type	Saddleback
ST segment	Gradually descending	Elevated ≥ 1mm

Quoted from: *Wilde AA, Antzelevitch C, Borggrefe M, et al. Proposed diagnostic criteria for the Brugada syndrome. Eur Heart J 2002 (uptodate.com)*

Electrophysiology

1) Coved type

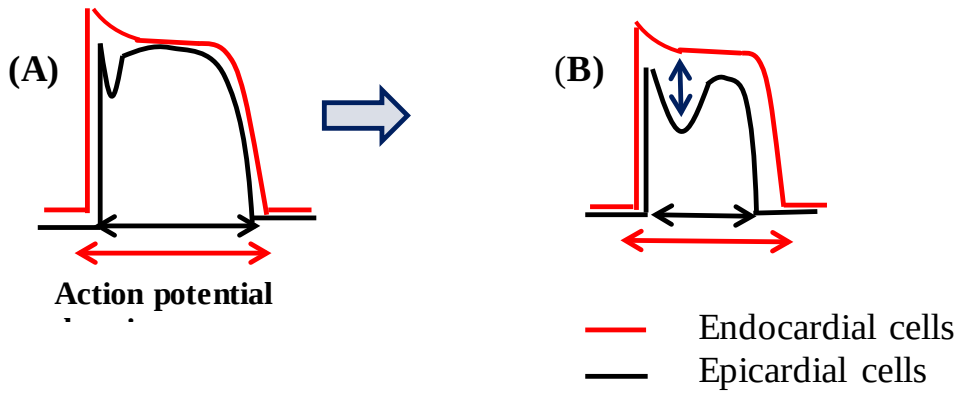
- Defect in Na channels in epicardial cells of right ventricular outlet tract (RVOT) exclusively leads to decreased Na influx . This leads to delayed conduction in this area than the rest of right ventricle .
- While right ventricle becomes depolarized , RVOT is still not activated (due to delayed conduction) so a current of injury passes from right ventricle to RVOT then to extracellular space towards right sided leads giving elevated ST segment.
- The repolarization process starts in right ventricle while the late RVOT gots depolarized. This creates current of injury moving from RVOT to right ventricle away from right sided leads giving inverted T wave.



Figures 6-9: Development of coved type
Quoted from *Meregalli, E. G. et al. Pathophysiological mechanisms of Brugada syndrome*

2) Saddleback type

- ü Normally epicardial cells are the first to be repolarized followed by myocardial then endocardial cells.This means while epicardial cells are reploarized, endocardial cells are still depolarized. This creates minimal voltage gradient between the two layers which is very low to form a reentry between them.
- ü A decrease in Na influx and Ca influx (found in Brugada) and normally prominent K efflux of epicardial cells lead to shortening of epicardial action potential duration and not endocardial.These effects will alter plateau phase mostly.
- ü During plateau phase, shortening of AP duration makes endocardium relatively depolarized compared to epicardium . This creates current of injury from endocardium to epicardium towards a lead causing ST elevation .
- ü As long as epicardial cells are still repolarizing firstly, the repolarization pathway is not altered so T wave is still upright.



Figures 6-10: Development of saddleback type

A: Action potential in both epicardial and endocardial layers are so close so voltage difference is so minimal to produce current **B:** shortening of action potential duration results in marked voltage gradient (blue arrow) between two layers producing current of injury responsible for ST elevation

Redrawn from *John Wiley and Sons, J. Cardiovasc. Electrophysiol.* ***12****, 268–272 (2001).*

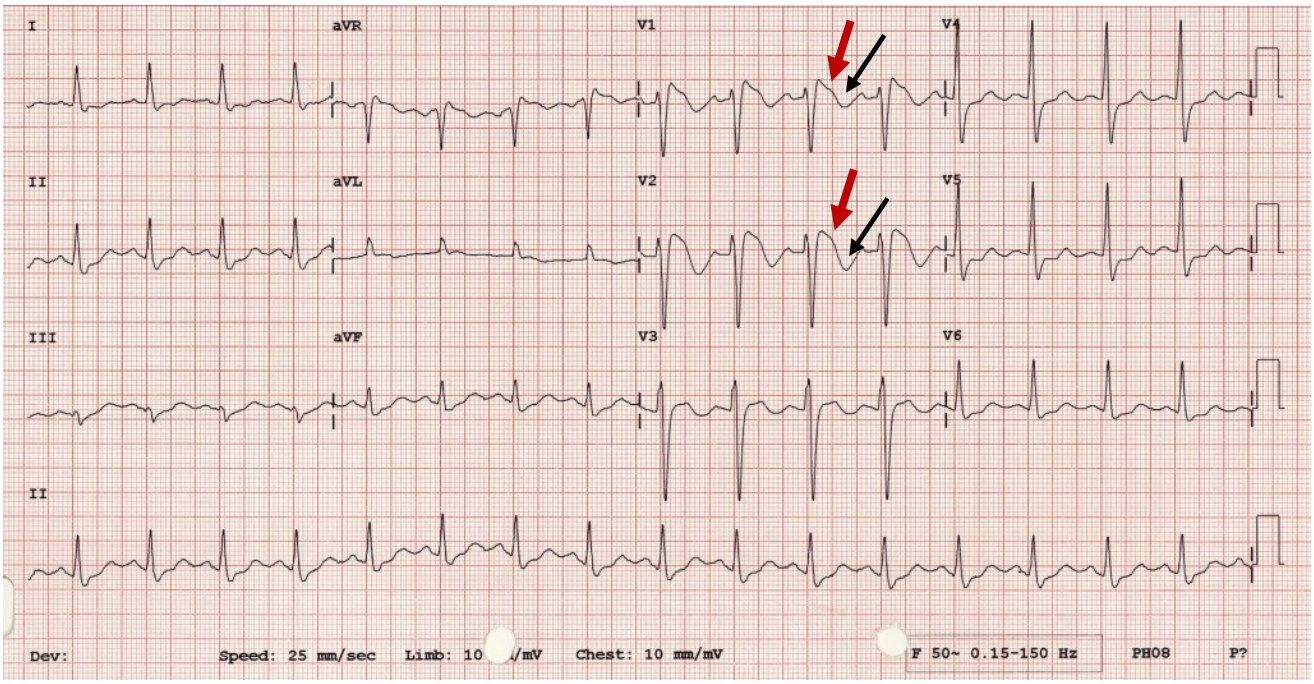


Figure 6-11: Brugada coved type

ST elevation (red arrow) and descends to inverted T wave (black arrow)

Quoted from *lifeinthefastlane.com*

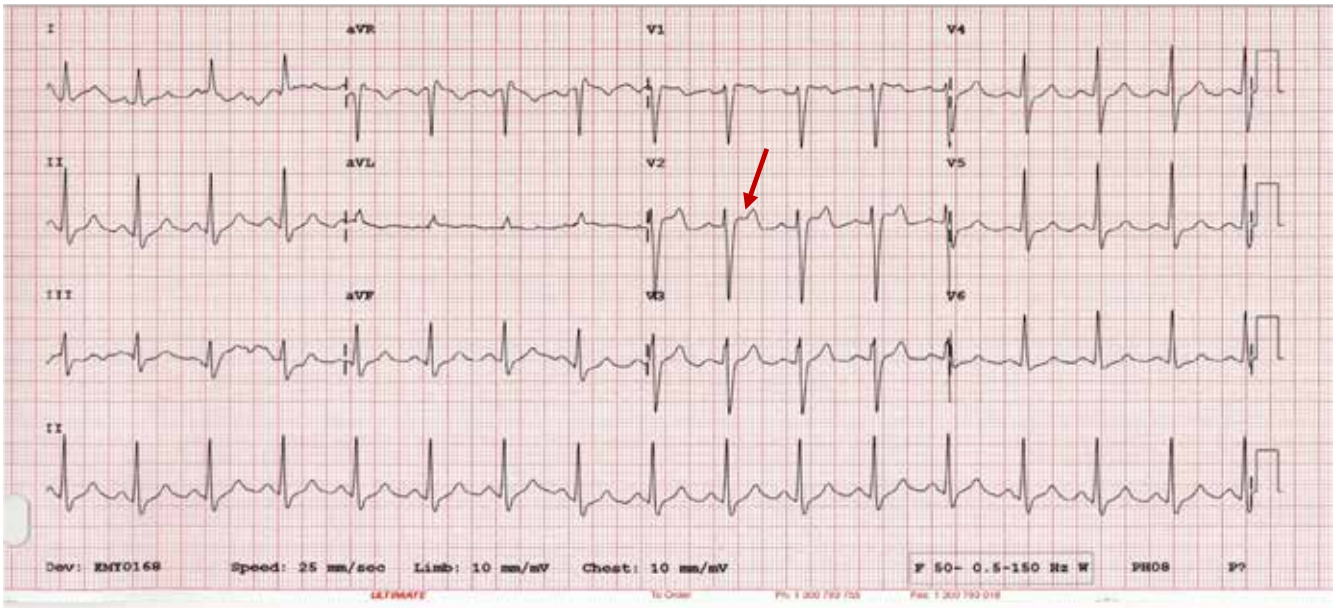


Figure 6-12: Brugada saddle back type

ST elevation, descends then ascends to upright T wave

Quoted from *dontforgetthebubbles.com/syncope-ecgs/*

Suspect Brugada if ST abnormalities are found in V1-V3 in young

5) Pericarditis

ü Anatomy overview :-

- The heart is composed of 3 layers (endocardium , myocardium and epicardium) . They are covered by serous sac called pericardium .
- Parietal pericardium is electrically inert so if it is involved solely , no ECG changes will be detected . The ECG changes associated with pericarditis occur as a result of concomitant inflammation of epicardium (the outermost layer of the heart).
- Sometimes , concomitant myocardial involvement occurs so totally it will result in perimyocarditis

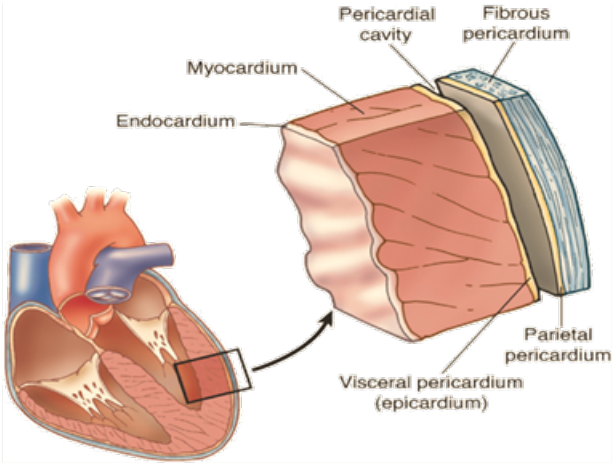


Figure 6-13: Heart and overlaying pericardium
Quoted from *http://anatomyofthefoot.com/4-layers-of-the-heart.html*

ECG features of pericarditis

ECG changes of pericarditis typically pass into 4 stages

Ø Stage 1 (first hours to days)

Diffuse concave ST elevation + PR segment depression in all leads
Reciprocal ST depression + PR segment elevation in aVR

Ø Stage 2 (first week) Normalization of ST and PR segment

Ø Stage 3 : Diffuse T wave inversions with isoelectric ST segment

Ø Stage 4 : either ECG return to normal or persistent T inversion denoting chronic pericarditis

Electrophysiology

1- Pericarditis leads to inflammation of adjacent epicardium (outermost layer of the heart) of both atrium and ventricle .
2- During systole (corresponds to ST segment) , current of injury moves from normal depoalrized endocardium to early partial repolarized epicardium causing diffuse ST elevation in all leads except right sided leads (aVR and V1) which have reciprocal ST depression.
3- Also current of injury moves from endocardium to epicardium during atrial systole (corresponds to PR segment) causing PR segment elevation in V1 and aVR with reciprocal PR depression in all other left sided chest leads and limb leads .

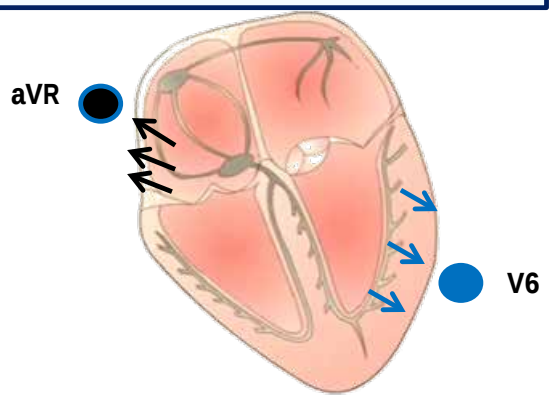


Figure 6-14: Current of injury in pericarditis
Blue arrows are current of injury in ventricle while black arrows are current of injury in atrium.
Quoted from *ECGteatcher.com*

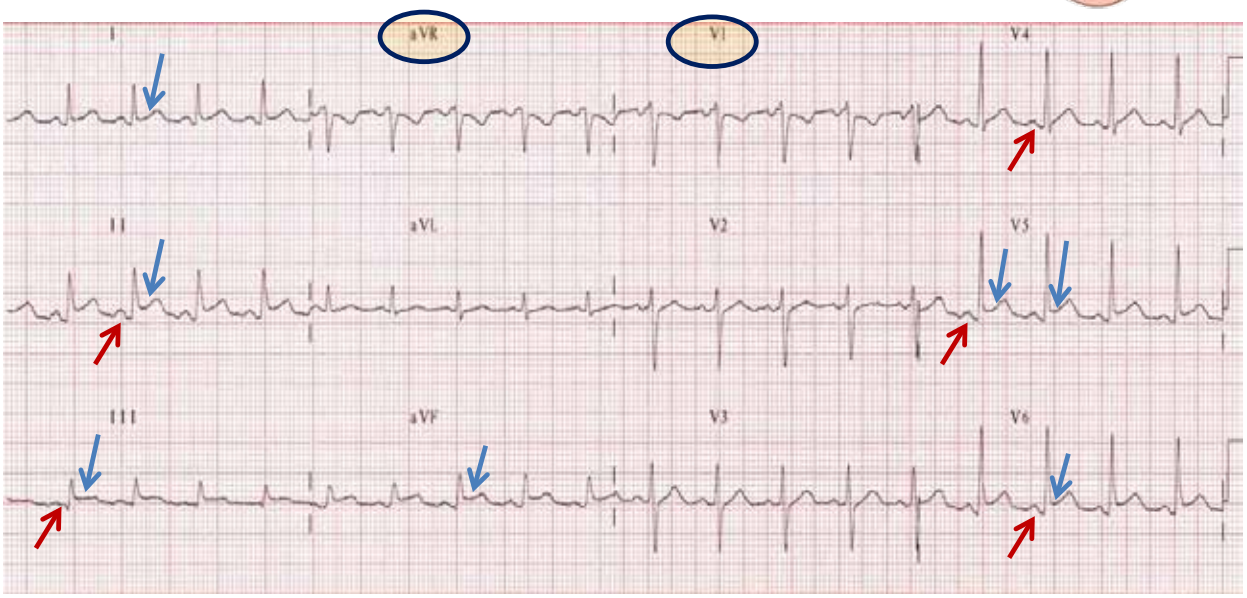


Figure 6-15: Pericarditis
Diffuse concave ST elevation (blue arrows) + PR depression (red arrows) Leads I, II, III, aVF and V4 to V6
Reciprocal ST depression and PR elevation in aVR and V1 marked by blue circles
There is also sinus tachycardia.
Quoted from *lifeinthefastlane.com*

6) Long QT interval

ü **Physiology**

- Gives information about the repolarization of the heart
- QT interval is inversely related to heart rate

Ø When heart rate is high, the repolarization time shortens thus QT is short

Ø When heart rate is low, the repolarization time prolongs thus QT is long

ü **Calculation of QT interval**

Ø **If heart rate (60 BPM):** apply QT duration directly

0.34- 0.44 sec (11 mm) width in males.
0.34- 0.46 sec (12 mm) width in females.

Ø **For any other heart rate:** $QT_C = QT / \sqrt{RR}$ (both in sec)

In the following Figure 6-16 is a part of patient ECG tracing. The device calculated QT interval being 0.45 sec which is normal.
HR is nearly around 55 (bradycardia) so by applying last equation

RR interval = 28 small box X 0.04 sec= 1.12 sec

QT interval = 19 small box X 0.04 sec= 0.76 sec

$QT_C = QT / RR^{1/3} = 0.76/1.04=$ **0.73 sec**

Very long QT interval, this patient has hypokalemia, hypocalcaemia and has received ceftriaxone and azithromycin for community acquired pneumonia. All of which lead to long QT interval.

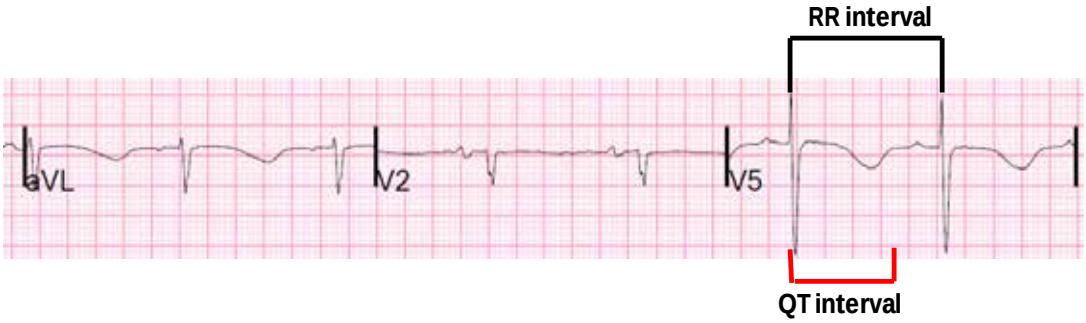


Figure 6-16: Long QT interval
Quoted from *hqmeded-ecg.blogspot.com.eg/2014/06/acquired-long-qt-do-not-trust.html*

Electrophysiology

1- Remember ions movement during phase 1 and 2 of action potential:

Phase 1 (early partial repolarization): this minor descent of the curve is formed by slow K efflux and inactivation of delayed Na current .

Phase 2 (plateau phase): this balance is caused by K efflux versus Ca influx .The net current is a repolarizing current.

2- Abnormalities that reduce net K efflux prolong repolarization and subsequently QT interval as :

- Ø Decreased K efflux by class IA (quinidine and procainamide) and class III (sotalol) antiarrythmic. They are K channel blockers.
- Ø Activation of delayed Na current during repolarization by ibutilide.

3- Normally repolarization of cardiac layers does not occur simultaneously. Delay in repolarization duration gives chance for any premature beat to be fired (triggered activity) and this beat may enters a reentry causing torsade de pointes (delayed repolarization means a layer will undergo repolarization & available for activation while the neighbouring one is still depolarized and and unavailable for activation creating media for reentry formation).

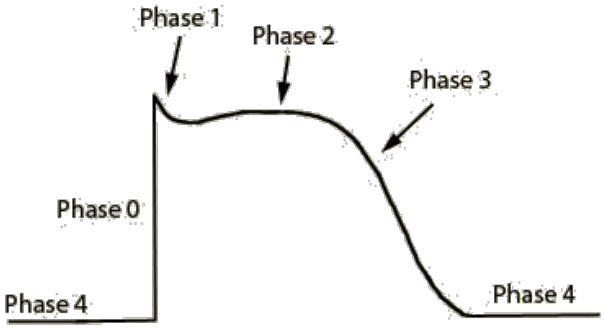


Figure 6-17: Action potential of the heart
Quoted from *theeplab.com/B-The-Members-Center/C-Cardiac-AnatomyPhysiology/*

Clinical pearls

1- Hypokalaemia is often associated with hypomagnesaemia, which increases the risk of malignant ventricular arrhythmias

2- Suspect hyperkalemia in any patient with a new bradyarrhythmia or AV block, especially patients with renal failure, on hemodialysis or taking any combination of ACE inhibitors, potassium-sparing diuretics and potassium supplements.

3- ECG changes do not correlate with serum levels and severity of hyperkalemia.

Chapter 7: Miscellaneous

Contents

1) Pulmonary embolism

ECG features

1- Sinus tachycardia (most common).
2- Right atrial enlargement and right ventricular enlargement (P-pulmonale and R/S ratio >1 (R larger than S) in V1.
3- $S_1Q_3T_3$ pattern in limb leads (deep S in LI , deep Q and inverted T in LIII).
4- Right ventricular strain pattern (inverted T and/or ST depression in V1 to V4).
5- Right axis deviation.
6- T wave inversion in inferior and anterior chest leads
   - ü Mostly noted in right sided leads LIII , V1 and V2.
   - ü Maximum magnitude of T invesrion in V1 and V2
   - ü These two findings together are highly sensetive 98 % and specific 92 % for Pulmonary embolism
7- Right bundle branch block

Electrophysiology

1- Occlusion of pulmonary artery by an embolus leads to right ventricular outlet obstruction so acute right ventricular and right atrial dilatation develops with subsequent right axis deviation.Pulmonary vascular resistence increases as embolism results in pulmonary vasoconstriction so pulmonary blood pressure will rise aggrevating enlargement of right sided chambers.
2- Marked right ventricular dilatation causes subendocardial ischemia which leads to RV straining .
3- Diminshed right ventricular output will result in hypoxia which is responsible for sinus tachycardia.
4- $S_1Q_3T_3$ pattern: as LIII is a right sided limb lead so RV straining appears as inverted T in it. Right axis deviation towards LIII causes prominent Q wave in LIII an prominent S wave in LI (as LI will be opposite LIII)
5- With increasing severity of enlargment , RV begins to enlarge to left side . This makes T inversion moves from LIII to aVF then LII and also from V1 ,V2 to V3 and may be V4.Another reason for concomitant anterior and inferior T wave inversion may be due to ischemia from low cardiac output which leads to underfilling of coronaries with worsening hypoxia.

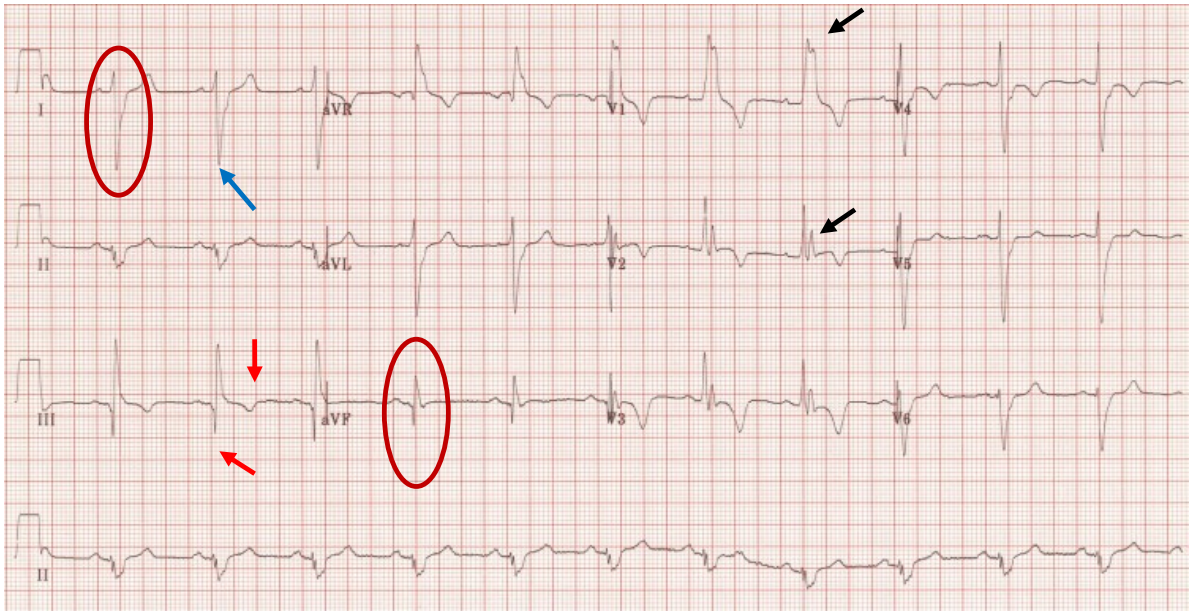


Figure 7-1: Pulmonary embolism

Right axis deviation (red circles) + S1Q3T3 (blue arrow, red arrows) + Right BBB pattern at V1 & V2 (black arrows for notched R) with inverted T waves in inferior & anterior leads

Quoted from *lifeinthefastlane.com*

2) Digitalis toxicity

ü Mechanism of action:

Digitalis blocks Na-K pump, this leads to enhancement of Na-Ca exchanger current with moving Na out of the cell and moving Ca to the inside of the cell.

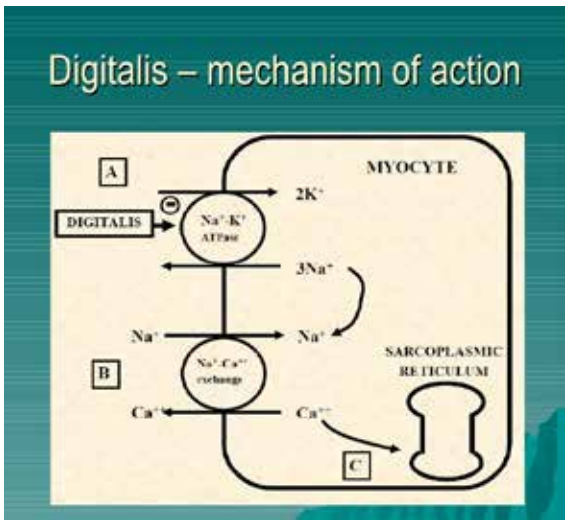


Figure 7-2: Mechanism of digitalis

Quoted from *Dhriti Brahma , NEIGRIHMS, shillong, india*

ü **Classic ECG features: -**

Supraventricular tachycardia + slow ventricular response

ü **Others include**

- Frequent PVCs (the most common abnormality), including ventricular bigeminy and trigeminy
- Sinus bradycardia or slow AF
- Any type of AV block (1st degree, 2nd degree & 3rd degree)
- Regularized AF = AF with complete heart block and a junctional or ventricular escape rhythm
- Ventricular tachycardia, including polymorphic and bidirectional.

Electrophysiology

1- Digitalis leads to increased Ca influx, this leads to increased automaticity and development of triggered activity responsible for premature beats and supraventricular tachycardia.

2- Digitalis causes increased K efflux and increases vagal tone . Both inhibits AVN conduction which leads to slow ventricular response.

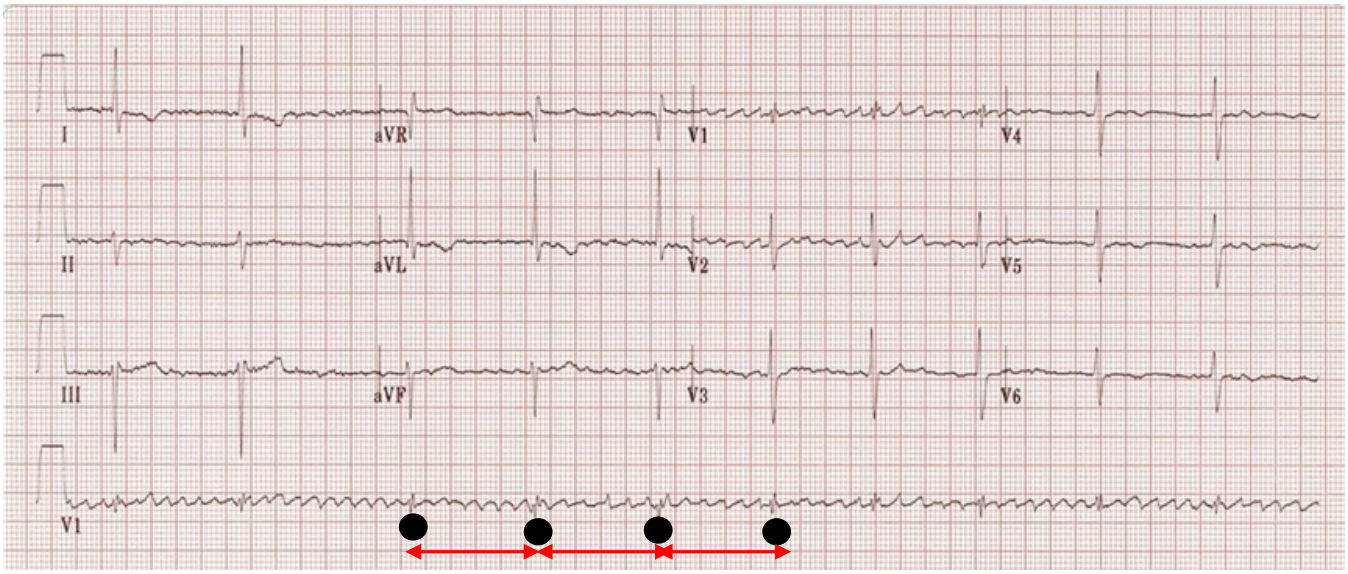


Figure 7-3: Digitalis toxicity
Many deflections preceding complexes (AF) + regular rhythm with slow rate 60 BPM (Junctional escape rhythm) + no relation between atrial and ventricular activity (3rd degree heart block)
Quoted from *lifeinthefastlane.com*

3) Hypothermia

Definition: temperature less than 34 Celsius degrees.

ECG features (mostly in V2-V5)

1- Prolongation of all intervals (PR, QT, RR) and QRS complex
2- Bradycardia
3- Osborn wave (prominent positive J wave)
4- Tachyarrhythmia like AF, VT and VF.
5- Asystole and cardiac arrest eventually.

Electrophysiology

1- Hypothermia causes slowing of impulse conduction through the heart this explains prolongation of intervals

2- Osborn wave is upward deflection at the site of J point (prominent J wave). It coincides with phase 1 of action potential (early partial repolarization) caused by K efflux.

Ø Normally repolarization starts in epicardium. Hypothermia causes slow K efflux with slow conduction so while the epicardial cells begins slowly to be partially repolarized; the endocardial cells are still depolarized. This voltage gradient generates current of injury passes from endocardium to epicardium towards lead during phase 1 causing upward deflection at J point known as Osborn (prominent J) wave.

N.B. Osborn wave is considered pathognomonic to hypothermia although found in hypercalcemia and other conditions.

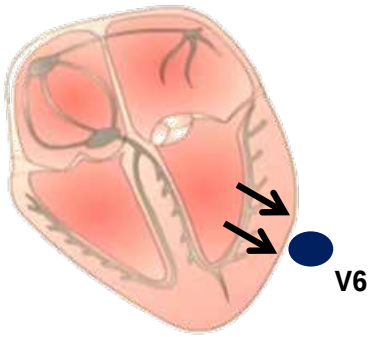


Figure 7-4: Osborn wave formation
Current passes from depolarized endocardium to partially repolarized epicardium forming upward deflection (Osborn wave)
Quoted from *ecgteacher.com*

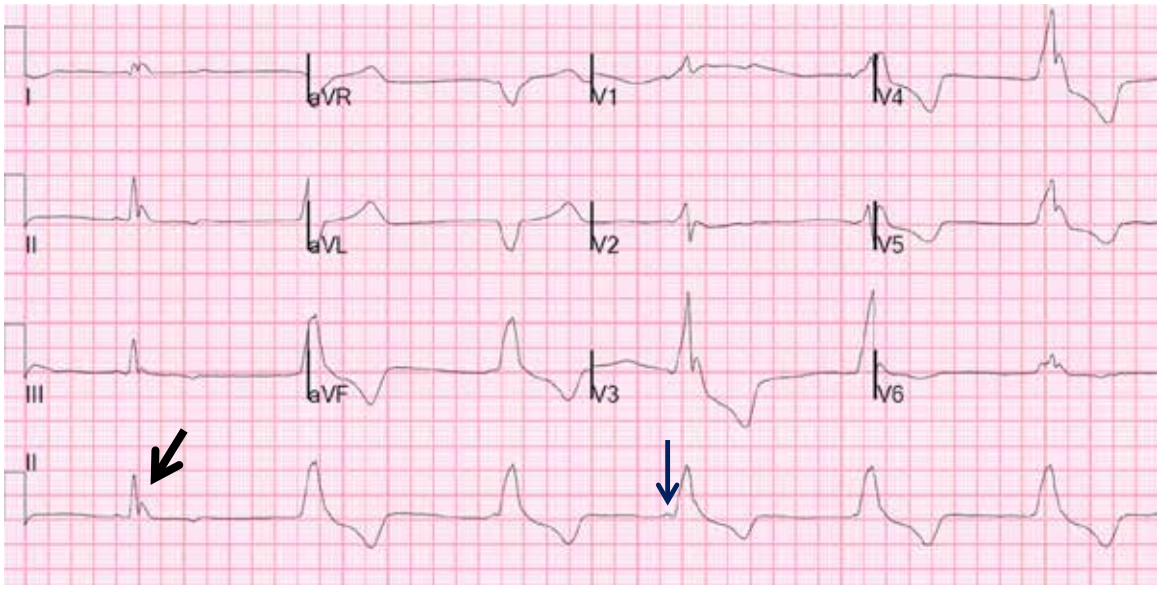


Figure 7-5: hypothermia

Patient's temp is 25.2 C; note Osborn waves (black arrow) on the first beat. Later beats are ventricular escape beats (wide complex + inverted T wave). Little P wave could be seen at 4th beat (blue arrow) (this P is not conducted below)

Quoted from *hqmeded-ecg.blogspot.com.eg/2015/01/massive-osborn-waves-of-severe.html*

4) Pacemaker rhythms

ü Artificial pacemakers are devices used to control the heart rate used for treatment of bradycardia they are 3 types of pacing :- atrial , ventricular and dual chamber pacing.

ü **Features of pacemaker rhythm**

1- Presence of spike usually 2mm lenght , the lenght of the spike becomes shorter when the lead is placed over epicardium (far from device).

2- Atrial pacing :- spike preceeds P waves which may appear normal

3- Ventricular pacing :- spike preceeds complex

- Right ventricular pacing gives complex of LBBB morphology
- Left ventricular pacing gives complex of RBBB morphology
- T wave is disconcordant with the QRS complex

4- Dual chamber pacing depends on the site of begining of pacing (may be atrial or ventricular or both).

5- Spikes may be not seen in all leads.

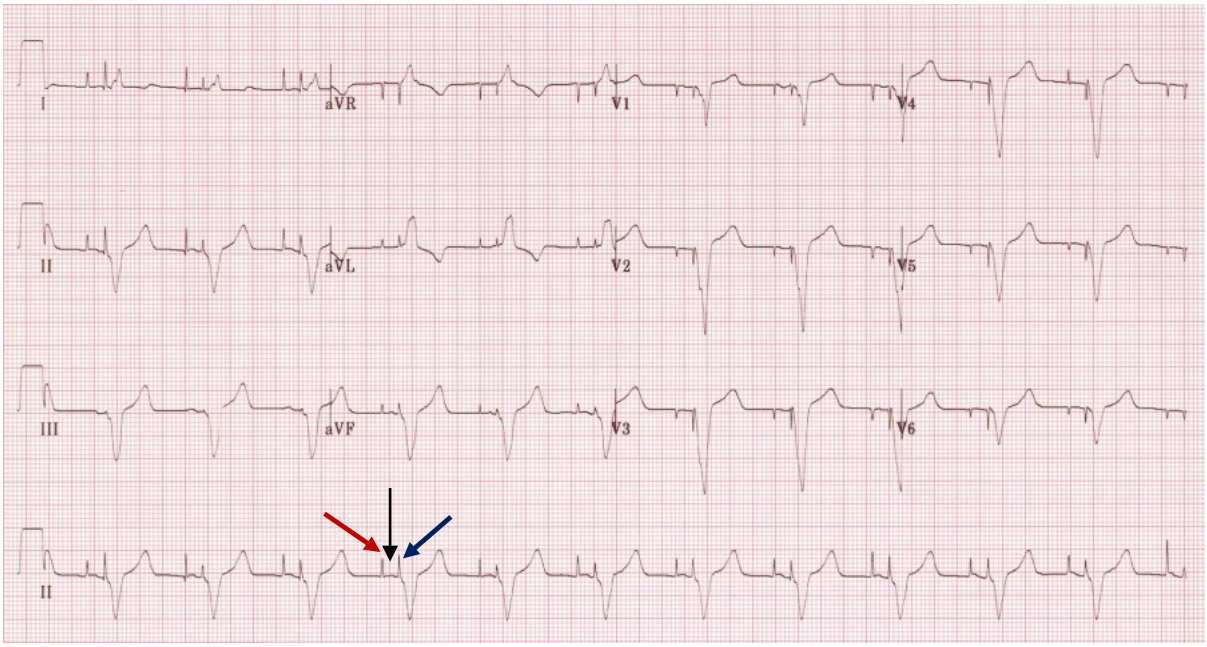


Figure 7-6: Dual chamber pacing
Red and blue arrows refer to spikes; black arrow refers to small P wave
Quoted from *lifeinthefastlane.com*

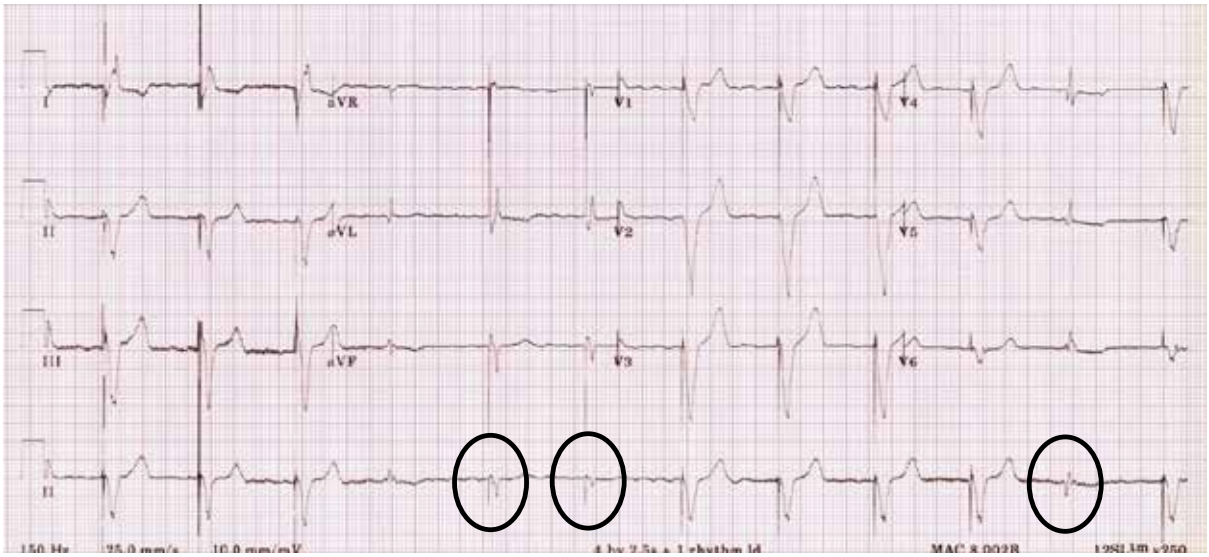


Figure 7-7: Ventricular pacing
Spikes before each complex. Note 3 circles represent complexes of different morphology (fusion complexes)
Quoted from *lifeinthefastlane.com*

5) Electrical alternans

ECG Features: alternating height of normally conducted QRS complex in any or all leads). It is commonly seen in pericardial effusion (with sinus tachycardia)

Electrophysiology:

1- Heart swings forwards and backwards with each beat during pericardial effusion. Swinging forwards makes the heart nearer to the chest wall and leads subsequently giving large complexes. When the heart gets backwards far from chest wall, the complexes appear smaller.

2- Cardiomegaly, left ventricular dysfunction and aortic regurge cause it.

NB. Another type of alternans called T wave alternans often seen in long QT interval. It is a warning sign of impending polymorphic ventricular tachycardia.

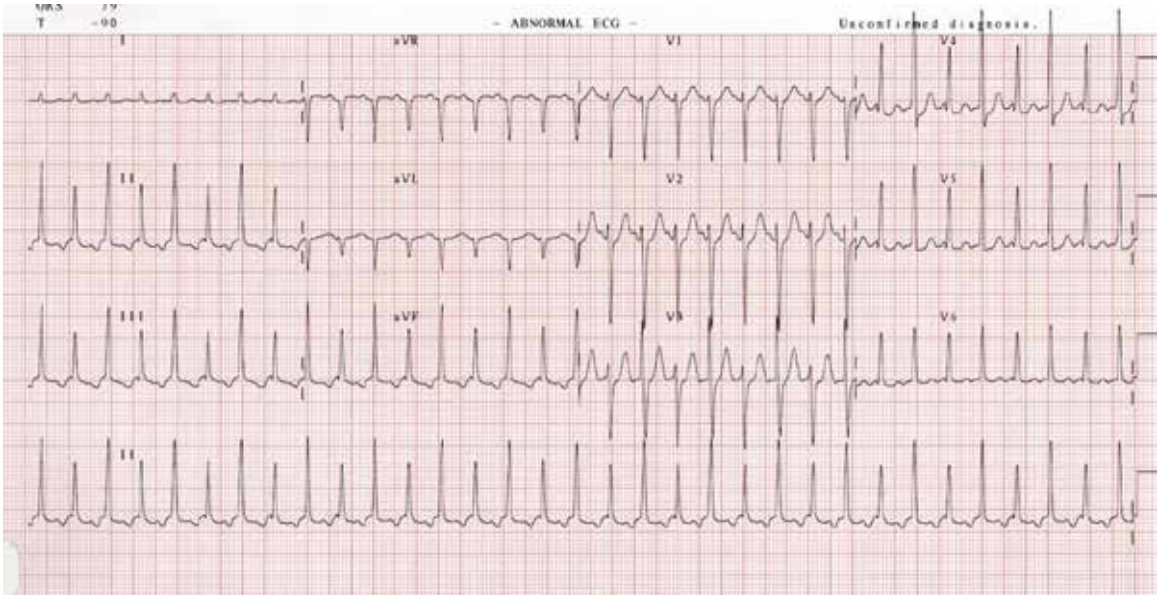


Figure 7-8: Pericardial effusion

QRS voltages are not constant (alternating high and low voltages; electrical alternans)
Note also tachycardia a frequent finding of pericardial effusion as well.
Quoted from *www.reddit.com/r/Pathognomonic/comments/21mwxj/electrical_alternans_pericardial_effusion/*

6) Motion artifact

They are irregularities on ECG tracing which may resemble some pathologies and makes ECG diagnosis is difficult. It is most often seen with tremors and shivering. Careful inspection of all leads and patient's history are needed in these situations to exclude or confirm it.

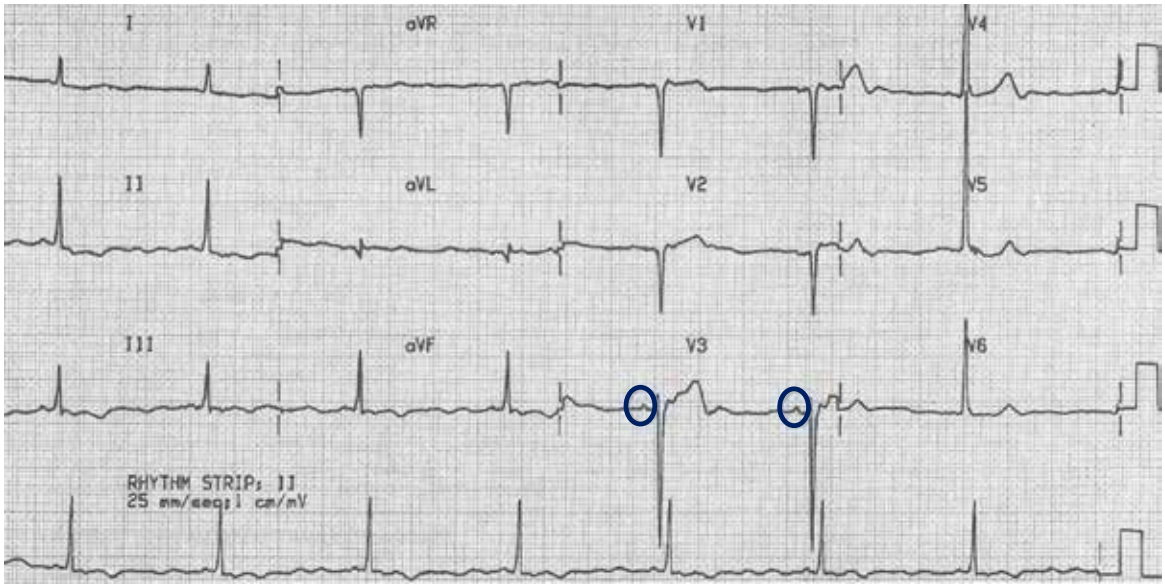


Figure 7-9: Tremors induced artifact with sinus bradycardia

The long strip gives the impression of atrial fibrillation. Bradycardia also gives the impression of complete heart block with AF.

Blue circles reveal two obvious well defined P waves. This is artifact and bradycardia in patient with Parkinsonism.

Quoted from *lifeinthefastlane.com/ecg-library/artefacts/*

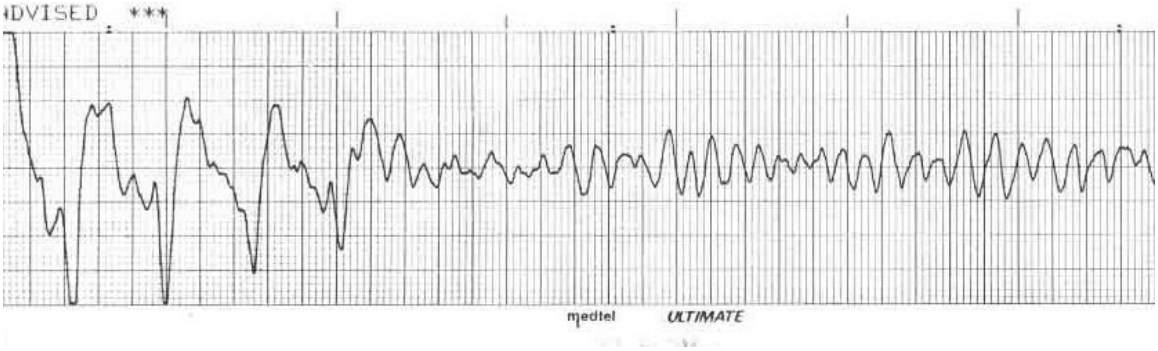


Figure 7-10: Artifact followed by ventricular fibrillation

This tracing is recorded while CPR efforts were done till the 2nd half revealed ventricular fibrillation. At this point resuscitating team stopped to reassess rhythm.

Quoted from *lifeinthefastlane.com/ecg-library/artefacts/*

Clinical pearls

1- Any new onset dysrhythmia in patient taking digitalis should raise susceptibility of digitalis toxicity.

2- Atrial flutter, atrial fibrillation and Mobitz II second degree heart block are the least likely abnormalities to occur with digitalis toxicity.

3- Check for K levels and related ECG changes in acute digitalis toxicity.

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